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OF

NEURO-SURGERY

AUTHORIZED BY THE SECRETARY OF WAR
UNDER SUPERVISION OF THE SURGEON GENERAL

Prepared under the direction of the Neuro-Surgical Section of the Division of Surgery, in collaboration with the Sections of Head Surgery, General Surgery, Orthopedic Surgery, and Neuro-Psychiatry, the Army Neuro-Surgical Laboratories, and the Instruction Laboratory of the Army Medical Museum

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PREFACE.

The Manual of Neuro-Surgery represents an effort to broaden the scope of War Surgery of the Nervous System, compiled in 1917. While the general aims of the 1917 volume have been followed in the Manual of Neuro-Surgery, the text has been amplified by the addition of discussions, from the neuro-surgical point of view, of the sympathetic system and its divisions, the ductless glands, the mastoid process, the accessory sinuses, and the laboratory methods of removing and preserving nerve tissue. More than 200 illustrations are used, many of them new, some of these being selected from moving-picture studies of nervous diseases.

The manuscript consists principally of original material prepared especially for this work, but extracts have been taken from the chapter in Keen's Surgery, "Fractures of the Skull," by Dr. Harvey Cushing; from "Equilibrium and Vertigo," by Dr. Isaac H. Jones; from the chapter on the technic of laminectomy, in "Surgery of the Spine and Spinal Cord," by Dr. Charles H. Frazier; and from the technic of the suboccipital operation in "Tumors of the Nervus Acusticus," by Dr. Harvey Cushing. Free use has been made of "Diagnosis of Nervous Diseases," by Purves Stewart; of Tinel's monograph on "Nerve Wounds"; and of the Proceedings of the Royal Society of Medicine, 1915–16, Volume X, part 3. Valuable illustrations have been reproduced from "Diseases of the Eye," by de Schweinitz.

Thus modified and amplified, the Manual of Neuro-Surgery is intended to meet the call for a comprehensive one-volume book of reference for students in the Army neuro-surgical schools in Philadelphia, New York, Chicago, and St. Louis, and for medical officers, in this country and overseas, who may be delegated to care for patients with neuro-surgical conditions.

Acknowledgment is made to the following publishers for helpful cooperation and for the privilege of using the chapters mentioned: William Wood & Co.; W. B. Saunders Co.; Lea & Febiger; D. Appleton & Co.; Edward Arnold; Bailliere, Tindal & Cox; Longmans, Green & Co.; and the Oxford Medical Publishers.

Thanks are due to William Wood & Co. for the privilege of using six color prints from "Diseases of the Eye," by Dr. Charles H. May.

This volume will not be offered for sale, its distribution being limited to the officers in the Medical Corps of the Army and to libraries receiving Government literature.

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MANUAL OF NEURO-SURGERY.

CHAPTER I.

ANATOMY AND PHYSIOLOGY OF THE BRAIN.

The nervous system consists of the brain, the spinal cord and the sympathetic system of plexuses, and the nerves connecting with the peripheral parts of the body and the internal organs.

To make a diagnosis of an organic nervous disease, each symptom should be interpreted by indicating a definite lesion of a certain part of the nerve structure. To do so it is necessary to have an accurate knowledge of the anatomy, physiology, and pathology of the central and peripheral nervous systems.

CEREBRUM.

The cerebrum is the principal part of the nervous system, and consists of two lateral hemispheres connected by a band of white fibers, the corpus callosum, and a system of ganglia, including the caudate and lenticular nuclei and the optic thalamus.

Each hemisphere is divided anatomically into a frontal, a parietal, a temporal, and an occipital lobe. (Figs. 1 and 2.)

The lobes are traversed by fissures which result in the formation of convolutions. The surface part of the convolution, called the cortex, is about one-eighth to one-fourth inch in thickness and consists of a system of nerve cells and fibers arranged in layers.

In the cortex are the so-called centers for every voluntary and involuntary movement, consciousness, and thought. Some of the fibers which rise from the cells of the cortex and pass through the internal capsule are called projection fibers and are concerned with motor, sensory, and special functions, while other fibers connecting one part of the brain with another are called association fibers and are concerned with the correlation of the functions of the different parts of the brain. A third or commissural system connects similar parts of the lateral hemisphere.

Within the brain is a system of communicating cavities or ventricles. These cavities are in continuation with the central cavity of

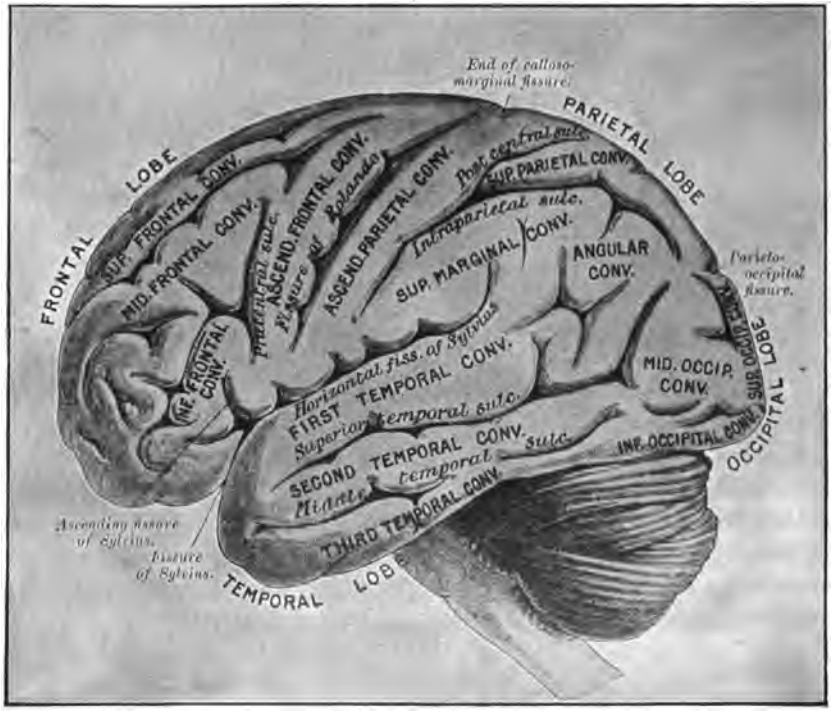

FIG. 1.—Convolutions and sulci on the external surface of the cerebral hemisphere. (Gray.)

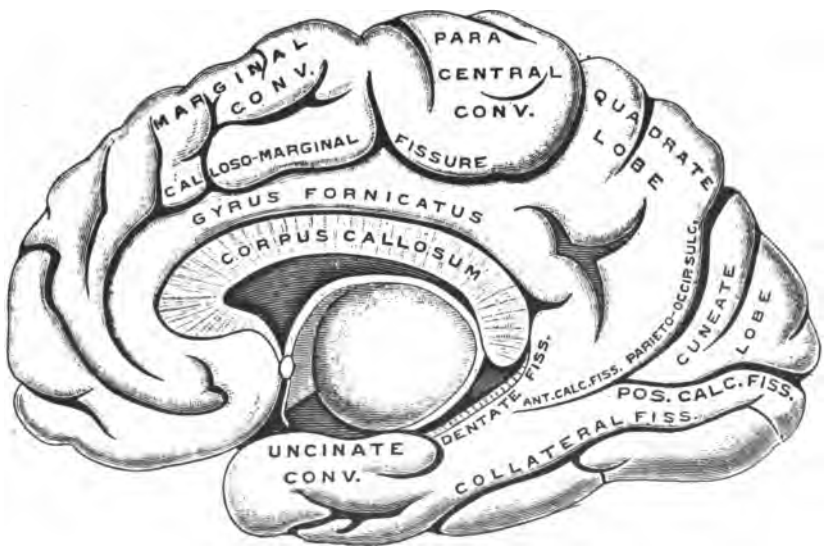

FIG. 2.—Convolutions and sulci on the mesial surface of the cerebral hemisphere. (Gray.)

the spinal cord. There are two lateral ventricles occupying parts of the cerebral hemispheres, the third ventricle, situated between the optic thalami, and the fourth, which is between the cerebellum and the medulla oblongata.

The brain is intimately surrounded by pia, which dips between the convolutions and into the fissures accompanying the vessel. Surrounding the pia is the dura-arachnoid, which envelops all parts of the brain and subdivides the cranial cavity into two parts—that

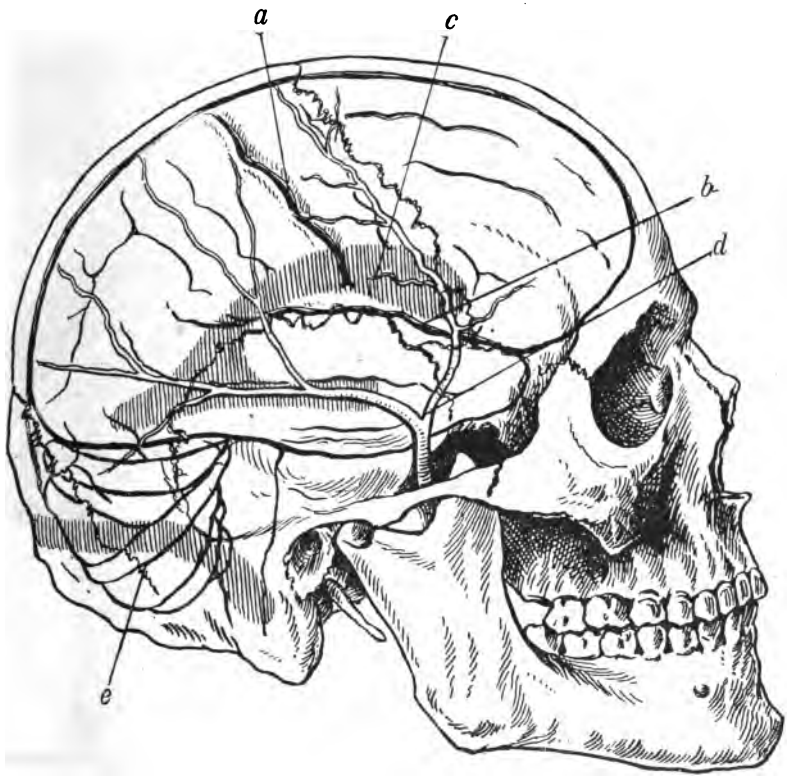

FIG. 3.—Topography of skull. *a*. Rolandic fissure. *b*. Sylvian fissure. *c*. Lateral ventricle. *d*. Middle meningeal artery with anterior and posterior divisions. *e*. Lateral sinus.

which includes the cerebrum proper, or the two lateral hemispheres, and the posterior part, which covers the cerebellum and is called the tentorium cerebelli.

CORTICAL LOCALIZATION.

As a result of investigation definite functions have been assigned to different parts of the cortex, these being *motor*, *sensory*, and *special*. The general underlying principle is that in the cortex are represented—as, for instance, in the motor—movement, and any irritation of a motor center will produce movement of the related part

in the opposite side of the body, while destruction will cause loss of that movement. The same principle is true of the other portions of the cortex. Not much is known of the function of the so-called central ganglia; that is, of the caudate and lenticular nuclei and the optic thalamus. The two cerebral hemispheres are in intimate connection with the cerebellum, which is the great coordinating center of all voluntary and involuntary movements.

Motor localization and motor symptomatology.—The motor centers have been placed entirely in front of the central or Rolandic fissure, in the pre-central and the anterior portion of the para-central convolutions. The para-central convolution is composed of the median

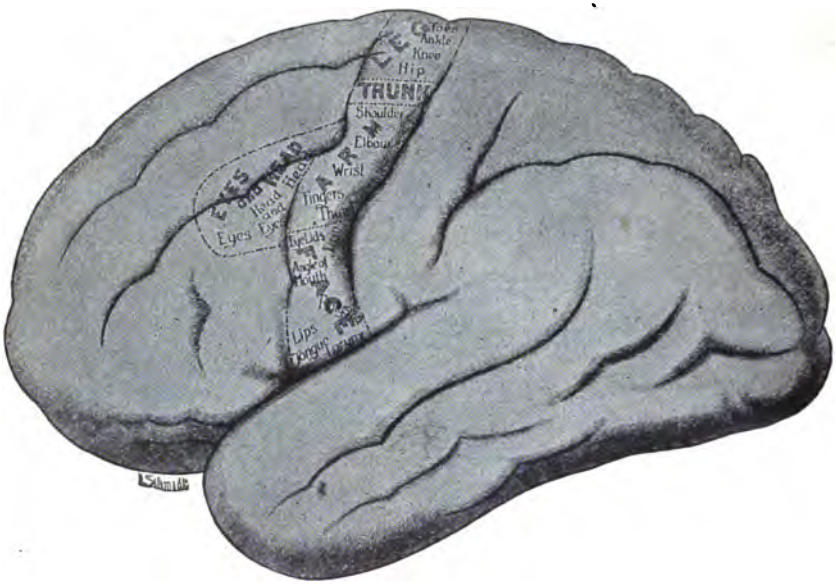

FIG. 4.—The subdivisions of the motor cortex. (Mills and Frazier.)

portion of the upper boundary of the pre- and post-central convolutions. The motor centers are subdivided into the head center, which is below, then come the centers for the arm, trunk, abdomen, and leg, the latter being in the topmost portion or in the para-central convolution.

It is important to understand that in the motor cortex is represented movement and not the action of an individual muscle. That being the case, in the arm center, for example, is located the movement of the thumb, fingers, elbow, shoulder, or any part of the upper limb. If there should be an irritation of any portion of the pre-central convolution, such as would be brought about by a bullet lying upon the pre-central convolution, there would result movements on the opposite side of the body, the beginning of the movement depend-

ing upon the part which is irritated. Such movements or convulsions are known as Jacksonian.

By *Jacksonian epilepsy*, therefore, is meant a convulsion which *always* begins in the same part of the body. If, for example, a con-

FIG. 5.—Jacksonian epilepsy in which the convulsion began in the right side of the face and extended to the right upper limb and was limited to these parts. Illustration shows the patient at the height of the convulsion. Abscess found in the middle and lower part of the pre-central convolution. (Weisenburg.)

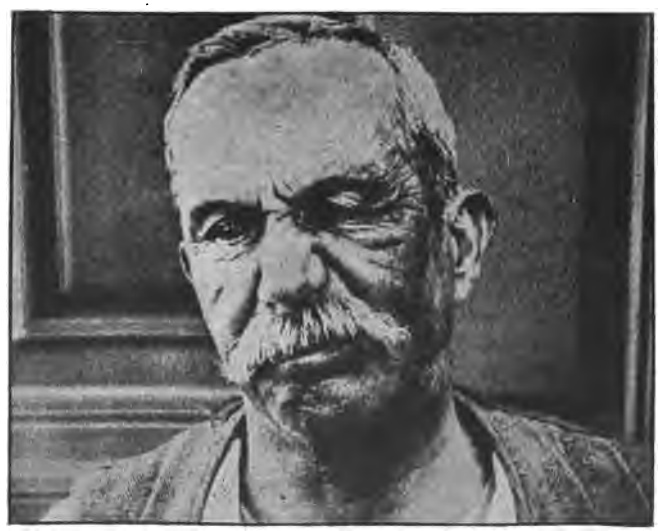

FIG. 6.—Jacksonian convulsion limited to the left face. (Weisenburg.)

vulsion should start in the fingers of the right hand and extend into the right shoulder and from there into the right face, it would be an indication that the origin of irritation was in the left pre-central

convolution, in the arm center, and that the lesion extended downward into the head center. If, on the other hand, the convulsion extended from the right arm into the right chest and right leg, it would indicate that the lesion extended from the middle or arm center upward into the leg center. It is important, therefore, to know where the convulsion begins and where it goes, for it is by such clinical symptoms that the surgeon is given the indication as to where to operate. Of course, a Jacksonian convulsion may at times extend and involve all parts of the body, but the important point is that it always begins in the same place. In Jacksonian epilepsy there need not be unconsciousness.

The *differential diagnosis* between the convulsions occurring in Jacksonian epilepsy and those occurring in idiopathic epilepsy and hysteria is very important.

FIG. 7.—Jacksonian convulsion which began with an outward movement of the right arm followed by a conjugate movement of the head and eyes to the right, and then an involvement of the whole of the right side. Illustration shows the right limbs in a tonic state, with head strongly deviated to the right. Note the position of the left leg, which is not involved. (Weisenburg.)

In *idiopathic epilepsy* there is no history of an antecedent period of good health followed by trauma, which is the case in Jacksonian epilepsy, for in idiopathic epilepsy the symptoms begin early in life and are progressive. The convulsions are nearly always preceded by an aura followed by a cry, the patient falling to the ground unconscious. The movements are never the same. They may begin in one arm in one attack, whereas in the succeeding attacks they may begin in both arms, for it is well known that epileptic attacks vary in their manifestation. In Jacksonian epilepsy there may or may not be unconsciousness, whereas in idiopathic epilepsy there is always loss or impairment of consciousness. In the Jacksonian variety the movements are nearly always tonic, with a well-marked clonic element, while in idiopathic epilepsy, although there may be a clonic succession, the movements are nearly always tonic. Besides, in idiopathic epilepsy the patient nearly always bites his tongue and voids urine, which is not the case in the Jacksonian type.

Again, in the epileptic there are present always other types of attack, such as *petit mal*, and of the psychic variety. In the *grand mal* or major attacks the movements are present in all parts of the body.

FIG. 8.—Illustration shows patient with idiopathic epilepsy. Note position of limbs in this illustration as well as in next, which shows the same patient. The attack was preceded by an aura followed by a cry, the patient falling to the ground unconscious. (Weisenburg.)

FIG. 9.—Showing patient with idiopathic epilepsy. Note position of limbs (see also fig. 8, which shows the same patient). Attack was preceded by an aura, followed by a cry, the patient falling to the ground unconscious. (Weisenburg.)

In *petit mal* they are only in part of the body and may consist only in champing of the lips or tongue or in falling. In the psychic variety the patient has an impairment or loss of consciousness which may last only for a second or so. There are besides so-called epileptic equivalents, in which the patient will not know what he is doing for varying lengths of time, lasting from a few minutes to many days. During this period he may apparently do normal things, yet after he regains consciousness he has no recollection of what happened. It is important to remember that in the well-marked epileptic there is always present more than one type of attack.

FIG. 10.—Idiopathic epilepsy, showing clonic phase, with frothing at the mouth. (Weisenburg.)

The convulsions occuring in *hysteria* or in the shell-shock cases are quite different, for here there is no loss of consciousness, although there may be psuedo-loss, and while the patient may bite his tongue he will not void urine, and the movements are altogether opposite in character to those found in the Jacksonian and idiopathic varieties; being wild or bizarre and neither tonic nor clonic. Besides, the history is quite different both in the onset of the attacks and the occurrence of other types of convulsions, and, as is well known, it is possible to suggest new symptoms to such patients.

It is important to distinguish the attacks occurring in *malingerers* from those occurring in the true hysterical or *shell-shock* patient, for in the latter the disease is just as distinct as Jacksonian epilepsy, while the malingering symptoms are altogether the result of the patient's cupidity. The convulsion of the malingerer can always be told from the organic type in the fact that it does not conform to the tonic clonic variety and differs from the hysterical attacks in the fact that there are not present any of the well-known stigmata or the

other symptoms of this disease. It is important, however, not to diagnose malingering until no other decision can be reached. All malingerers should be examined for mental deficiency.

Destructive cortical motor lesions.—It is possible to recognize at once that an irritating lesion has become destructive by the appearance of permanent impairment of movement. If, for example, there should be a lesion of the thumb center there would be impairment of movement of the thumb, but such isolated paralyses hardly ever occur. Rarely a monoplegia of an arm or leg may be present, but this is not the rule. A destructive lesion of the entire pre-central convolution on one side will cause a hemiplegia on the other side of the body.

Hemiplegia.—By hemiplegia is meant an impairment in the movement of a leg, abdomen, chest, arm, and the lower part of the face on one side of the body. It is well-known that in hemiplegia there is an escape of certain movements, such as the wrinkling of the forehead, moving the eyeballs, talking, eating, swallowing, and breathing. The proper performance of any of these functions necessitates the action of the muscles of both sides of the body, for it is apparent that the normal person can not wrinkle one side of the forehead, move one eyeball, talk, eat, swallow or breathe on one side of the body. Therefore these so-called bilateral functions have a representation in both cerebral hemispheres. While an irritation of any one of these centers—for example, the head and eye center—will cause a movement of the head and eyes to the opposite side, yet a destruction of one of these centers will not cause a permanent impairment of these functions, for the other side of the brain is capable of controlling such functions. While it is undoubtedly true that in freshly developed hemiplegia there is impairment of the bilaterally acting functions for a short period, yet such impairment is never persistent, excepting perhaps to a minor degree.

It is important to recognize the *symptoms of fresh hemiplegia* such as would result from an injury to the head or from hemorrhage. In a frank hemiplegia if the lesion is of any size, the patient is always unconscious. All the limbs are flaccid, and at first all the reflexes are lost. On inspection, however, it will be seen that there will be a flattening of the cheek on the paralyzed side, dribbling of saliva on this side, and in the stertorous respiration which nearly always is present, the paralyzed cheek will flap in and out with inspiration and expiration. Besides, the umbilical reflex is always absent on this side. The Babinski reflex becomes apparent within a few hours after the appearance of the hemiplegia. With this there is nearly always return of tonicity in all of the limbs, particularly on the paralyzed side, and there will be a return of reflexes, especially on the involved side. Consciousness will return, depending upon the size of the lesion, usually in from a few minutes to an hour or two.

If consciousness does not return in 24 hours the outlook is hopeless. The earlier the return the better the prognosis.

With the return of power, which can be noticed in some cases immediately and in others not until hours, days, or weeks later, there is a greater return of power in the flexor group of muscles in the upper limb, and in the extensor group in the lower limb. This unequal return causes a flexor contracture in the upper limb and an extensor contracture in the lower limb. The reason for this unequal return of power is the greater development of the flexor muscles in the upper limb and the extensor group in the lower limb. In a well-marked hemiplegia, then, there are present the following groups of symptoms: Impairment of power in the leg, arm, lower part of the face; the limbs are spastic and contractured; the reflexes are increased; the Babinski phenomenon is present.

Centers for conjugate deviation of the head and eyes.—In the middle of the second frontal convolution, just anterior to the pre-central convolution, is the motor cortical center for the deviation of the head and eyes. There is a separate center for the movement of the head and for the eyes, but inasmuch as the head and eyes usually act in unison, their centers are in practically the same area. Conjugate deviation of the head and eyes is a very common symptom in destructive and especially in irritative lesions of the brain. It need not and does not indicate a lesion in a particular part of the brain, for lesions in any part of the brain may cause this symptom. Its importance lies in the fact that its presence at once indicates a lesion in the cerebral hemispheres.

By conjugate deviation is meant a deviation of the head and eyes to one side. In conjugate deviation of the head and eyes to the right both the head and eyes are deviated strongly to that side and remain fixed in that position. Usually such patients are unconscious, but if consciousness should be regained they would be unable to move either the head or eyes from the position of extreme deviation.

Conjugate deviation may be either irritative or destructive. In an *irritative* lesion of the right side the head and eyes will be deviated to the left. Similarly an irritative lesion of the left side of the brain will cause deviation to the right. The irritative symptoms can be recognized from the destructive by the fact that the eyes will be in a spasm or will move to and fro to the side opposite the lesion. In a *destructive* lesion the head and eyes remain fixed. Besides, in irritative lesions there are nearly always Jacksonian convulsions on the side to which the head and eyes deviate, whereas in destructive lesions there is usually an accompanying hemiplegia.

It is important to recognize whether the lesion is irritative or destructive, for in *irritative* lesions the head looks away from the lesion, whereas in those which are *destructive* the head and eyes look

toward the lesion. This is because the function of the right head and eye center is to deviate these structures to the left. The function of the left is to deviate the head and eyes to the right. An irritation of one center will cause a deviation to the opposite side, but if there should be, for example, a destruction of the head and eye center on the right side the left center, because of its unopposed action, would deviate the head and eyes to the paralyzed side.

Conjugate deviation of the head and eyes must be distinguished from *paralysis of associated ocular movement.* By paralysis of associated ocular movement to the right, for example, is meant that the patient is unable to look to the right with either eyeball but he is able to look up and down and to the left. Such a patient is nearly always conscious, and there is no spasm of the ocular muscles. Such a condition always indicates a lesion of the posterior longitudinal bundle in the pons.

Method of obtaining motor symptoms and their interpretation.—Every part of the body has a nuclear representation in the nervous system. By that is meant that every muscle-fiber is in relation with cells which are located in the anterior horns of the spinal cord throughout its whole extent, and in the motor nuclei of the different cranial nerves from the third to the twelfth, inclusive, in the crus, pons and medulla, these parts being really the upward continuation of the spinal cord. Thus, for instance, if there is a destruction of the nerve cells in the facial nuclei in the pons, there will be loss of function in the muscles of the face; and if there is a disturbance of the cells in the anterior portions of the spinal cord in the lumbar region, there will be alteration of function in the corresponding muscles of the leg.

In other words, there are two great forms of representation in the nervous system, that in the *cortex*, which is concerned purely with *motion*, while in the *spinal cord* and its prolongation, including in this the crus, pons, and medulla, are represented not motion, but the innervation of the individual muscle-fibers. There must necessarily be a connection between these two systems, and this is affected by means of the motor tracts. These have their origin in the motor centers in the cortex. From here they go through the anterior third of the posterior limb of the internal capsule, and those fibers which are concerned with the movements of the opposite side of the body below the head go through the crus, pons, cross or decussate in the medulla, and then are transmitted by means of the crossed pyramidal tracts into the spinal cord. Some of these fibers do not decussate in the medulla, but descend on the same side of the cord in the direct pyramidal tract. From here these fibers go to the cells in the anterior horn. For example, the nerve-fibers which come from the nerve-cells in the leg center first go through the internal capsule, then the crus, pons, cross over in the medulla, then go

through the crossed pyramidal tract all the way down the cord to the lumbar segment, and then join the cells in the anterior horn; while the fibers in association with the arm go only as far as the cells in the anterior horn of the cervical part of the cord. Correspondingly, the motor fibers which come from the face center go through the internal capsule and leave the motor tracts in the upper part of the pons, because the facial nuclei are in the lower part of the pons, while those fibers which are in relation with the muscles of the eyeball begin to leave the motor columns just below the internal capsule and above the crus, because the oculomotor nuclei are in the crus and pons.

The upper and lower motor neurons or systems.—The upper part of the motor system, that is, the cortical cells and the fibers from them to the nuclei of the motor cranial nerves in the crus, pons and medulla, or to the cells in the anterior horns of the spinal cord, but not including them, is called the upper motor neuron or system. By the lower motor neuron or system is meant the nuclei of the motor cranial nerves or of the cells of the anterior horn of the spinal cord and the fibers coming from them, including the motor peripheral or cranial nerves as far as their ending in the periphery. We see, then, that each neuron or system, so-called, has its own center or nerve-cell and a fiber continuation.

A normal relation between the upper and lower motor neurons or systems is necessary in the performance of any movement, for, while the impulse originates in the upper neuron, its performance is the result of the action of the lower, and if there is a lesion in either there necessarily results a disturbance of this relation or tone. Thus, in a lesion of the upper neuron there will be overaction of the lower, and vice versa. This overaction on the part of the lower motor neuron is manifested by an exaggeration of the tendon reflexes and spasticity or stiffness in movement, and on the part of the upper by a flaccidity or loss of tone and abolition of reflexes. In other words, in a lesion in the upper motor neuron there will be spasticity and increase in the tendon reflexes, while in a lesion of the lower, flaccidity and loss of the tendon reflexes.

Reflexes.—It is, then, upon the normal relation of the tone between the upper and lower motor neurons or systems that the condition of the tendon reflexes will depend. Every reflex has its physiologic arc, this consisting of a sensory impulse, a center, and a motor response. The simplest example is the knee or patellar jerk, in which, after tapping the patellar tendon, the impulse is carried by the sensory nerves and posterior roots to the cells of the anterior horn in the second, third, and fourth lumbar segments, and from here the motor ponse is transmitted by the anterior roots and the peripheral nerves. If there is a lesion in any portion of this arc, there

will be loss of the reflex, no matter what the condition above in the spinal cord and brain. The first principle, then, in the attainment of any reflex is to have its arc intact, and in normal condition. Should there be a lesion, however, in any portion of the upper motor neuron or system anywhere in its course, thus disturbing the normal tone, there will result exaggeration of the reflexes because of loss of cerebral, or what has often been called inhibitory, influence. That every reflex has a cerebral influence is proved by the fact that if there is a complete transverse lesion, for instance, in one segment of the cervical cord, all the reflexes below are lost, even though the arcs are intact.

FIG. 11.—Method of obtaining knee jerk, showing position of legs and manner of tapping. (Weisenburg.)

Reflexes are of two kinds, first, deep or tendon, and, second, superficial or skin. The usual tendon reflexes are:

(*a*) In the upper limb; the biceps and triceps.

The *Biceps reflex* is obtained by having the patient flex the arm on the forearm, at right angles, with the thumb of one hand of the examiner on the biceps tendon. Striking the thumb with the percussion hammer will produce flexion of the forearm upon the arm. The center of the reflex is in the fifth cervical segment.

Triceps reflex.—With the arm in the same position, if the triceps tendon is struck near its insertion in the elbow, extension of the forearm on the arm will result. Spinal center is in the sixth and seventh cervical segments.

(*b*) In the lower limb, the *knee or patellar jerk* is best obtained by extending the leg upon the thigh at an angle of about 45°, the patient being in a sitting position. The examiner should then place one hand over the quadriceps muscle, striking the patellar tendon near its insertion with the other hand, there resulting a forward movement of the leg upon the thigh. Even a slight contraction of the quadriceps can thus be appreciated. The spinal center is in the second, third, and fourth lumbar. When the reflex is diminished, it may sometimes be brought out by reinforcement. This is done by having the patient lock his hand and then pull them apart, the examiner striking the tendon at the time of the greatest effort.

FIG. 12.—Method of obtaining ankle clonus. The leg should always be bent at the knee. Clonus is obtained by a sudden flexion of the foot on the leg. (Weisenburg.)

Achilles jerk is best obtained by having the patient kneel on a chair and then tapping the Achilles tendon near its insertion into the heel. A flexion of the foot on the leg will result. Spinal center is in the first sacral.

Ankle and patellar clonus.—A clonus is obtained only when there is an exaggerated tonicity, and always indicates a lesion of the motor or pyramidal tracts. Ankle clonus is obtained by first flexing the leg on the thigh. With one hand held over the calf of the leg, the other holding the foot, a sudden flexion of the foot on the leg
le, this resulting in to-and-fro movements which are regular
thm.

Patellar clonus.—With the leg extended on the thigh the patella is grasped between the thumb and forefinger and suddenly brought forward.

Biceps and triceps clonus is sometimes obtained similarly to that of the ordinary reflexes.

Rarely *ankle clonus* and sometimes *patellar clonus* can be obtained in *hysteria* or shell-shock, but the movements are not regular and the rhythm is influenced by the will.

Skin or superficial reflexes.—The *abdominal* or umbilicus reflex is obtained by stroking on one side of the abdomen, the umbilicus moving toward the side stroked. Spinal center is in the ninth, tenth, and eleventh thoracic segments.

The *cremasteric* reflex is obtained by irritating the inner portion of the upper thigh, this resulting in upward movement of the scrotum. Spinal center is in the first lumbar segment.

The *plantar reflex* is obtained by irritating the plantar surface of the foot, flexion of the toes resulting. Spinal center is in the second sacral. The *Babinski reflex* is obtained by irritating the plantar surface, extension of the toes resulting. Irritation is best produced with a match, which should first be drawn along the outer part of the foot and then across the sole. The important part of this reflex is the extension of the large toe, and the movements of the small toes may be disregarded. It is advisable to first grasp the foot at the ankle so as to prevent any voluntary movement. This reflex is never obtained in a functional condition and is always indicative of a lesion of those motor fibers which are in relation with the leg. A lesion of the motor fibers in relation with the upper limb will not produce this reflex.

Impairment of power.—The nature of the motor loss will also differ in lesions of the two neurons or systems. In a lesion of the upper, the paralysis will be that of movement. For example, should there be a lesion of the fibers in relation with the arm, there will be total loss of movement in the arm, while in a lesion of the lower neuron, the extent of the paralysis will depend upon the degree of involvement either of the nerve cells or of the motor fibers innervating the arm. In the former there will be an exaggeration of tone, or spasticity with increased reflexes; in the latter, loss of tone, or flaccidity, with loss of reflexes. Besides, in the latter condition, as the cells of the anterior horn are trophic in function, there will be atrophy of the muscles and electrical reactions of degeneration. Disturbances in vasomotor function occur in both. It must not be forgotten, however, that the paralyzed limbs in an upper neuron lesion may be flaccid for a number of hours and rarely for a day or more in severe destructive conditions.

Motor speech centers.—In right-handed individuals the speech centers are located in the left side of the brain and in the left-handed in the right side. As most of us are right-handed the left side of the brain is preponderantly developed. The motor and sensory centers are located chiefly in Broca's convolution and the temporal lobes. These areas on the right side of the brain are in the average right-handed individual undeveloped and are known as the silent area, lesions of which give no definite symptoms.

It is important, therefore, to determine either the right- or left-handedness of the individual. If a person uses both hands equally and yet writes with the left hand, he is left-handed, for it is the function of writing which determines on which side of the brain the speech centers are located. This can at once be appreciated when the importance of writing in the development of language is considered. Most of us learn to speak both by writing and word hearing. Writing, therefore, at once assumes a very important place in the mental development of the individual, much more so than any skilful hand manipulation, for writing not only is developed earlier in the life of an individual, but is concerned with more intellectual functions.

The motor speech centers are located in the so-called Broca's convolution, it being around the end of the anterior limb of the fissure of Sylvius, in the left side of the brain in right-handed individuals. Broca's convolution is anterior to the inferior portion of the precentral convolution or the head center. Lesions of this area cause motor aphasia.

Symptoms of motor aphasia.—In motor aphasia a person knows what he wants to say, appreciates what is said to him, knows the meaning of written words and letters and can read, but his difficulty consists in the fact that he is unable to express his thoughts in spoken words. The extent of the aphasia varies, depending upon the completeness of the lesion. In complete motor aphasia there is inability to utter a single word, while in the incomplete form the patient may be able to talk somewhat.

This inability to express in spoken words what an individual desires to say is not the result of paralysis of the lips, tongue, larynx, or vocal cords, for these parts can be used for any other purpose. It is the result of the impairment of the particular center which coordinates or which controls the function of expressing thoughts in spoken words.

Motor agraphia.—Usually in association with motor aphasia there is motor agraphia, by which is meant inability to express thoughts in ——tten words or letters. From what has been said of the develop-
f right- or left-handedness, it at once becomes apparent that
:h as language is acquired largely through writing, loss of

ability to express one's thoughts in spoken words is exactly in proportion to the loss of ability to express one's self in written words.

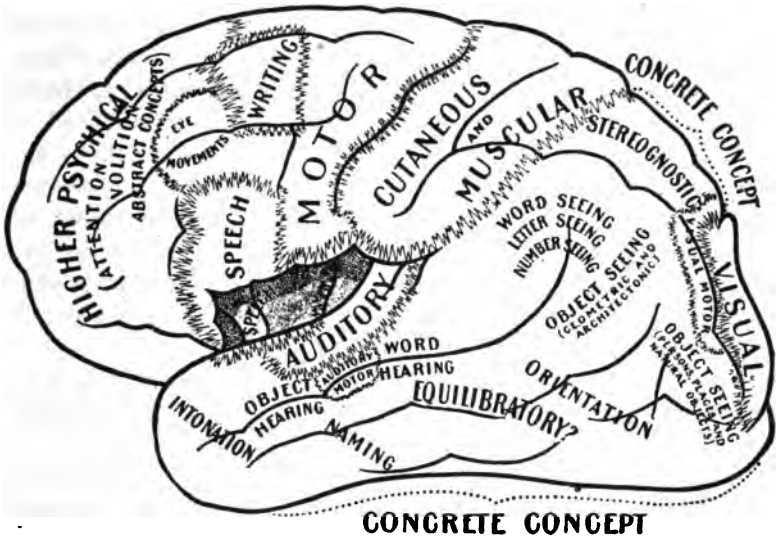

FIG. 13.—Side view of human brain, showing localization of functions. (Charles K. Mills.)

Taking advantage of such knowledge the re-education of motor aphasics is largely brought about through writing, for as one ac-

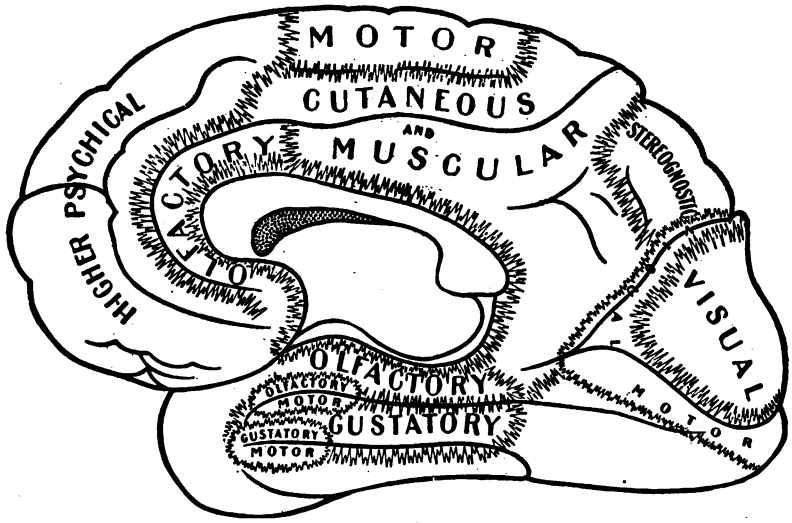

FIG. 14.—View of the mesial surface of the human brain, showing localization of functions. (Charles K. Mills.)

quires the ability to write a word, the ability to speak such a word is also attained.

Sensory localization.—The arrangement of the sensory fibers is more complex than that of the motor because of the great number and variety of sensations. The principle, however, is the same. There is still a great lack of definite knowledge regarding the course of the sensory fibers. It must be remembered that while motor impulses travel from the cortex, sensory impulses are transmitted to the cortex by means of the peripheral sensory nerves and the spinal cord. It is possible that in the cortex sensation is represented similarly to that of motion, and that just as in the motor area there is representation of motion, so in the sensory centers there is representation of the sensation in the particular movement, and that there is a correlation of function between the two. In support of that theory is the fact that the cortical motor and sensory centers concerned in a particular movement are in apposition.

Within a few years our views of the subdivisions of sensation have been considerably modified, chiefly through the work of Head, in association with Sherren and Rivers. Instead of the usually accepted subdivisions of touch, pain, and heat and cold, these authors have shown that common sensation is based upon three kinds of sensibility which are present and may be demonstrated in the peripheral system.

"(1) A system corresponding to the group of impulses which they have called *deep sensibility*. The end-organs of this system respond to the stimulus of pressure and to the movement of joints, tendons, and muscles. Painful impulses can also arise within this system in consequence of injury of a joint or excessive pressure. This sensory mechanism is capable of responding in such a way that the patient appreciates both the locality of the stimulus and the direction of movement in any joint which lies within an area innervated solely by this system; and yet the integrity of deep sensibility carries with it no power of appreciating a stimulus, such as that of cotton-wool even over hairy parts. Nor does it permit of the discrimination of two compass-points applied simultaneously to the skin, even when widely separated.

"The fibers which connect these sensory impulses run mainly with the muscular nerves, and are not destroyed by division of all the sensory nerves to the skin.

"(2) *The protopathic system*, capable of responding to painful cutaneous stimuli and to the more extremes of heat and cold. Its end-organs are grouped in points on the surface of the body, sensitive to one only of these stimuli. Their response is diffuse, and unaccompanied by any definite appreciation of the locality of the spot stimulated.

"(3) *The epicritic system.*—To the impulses of this system we the power of cutaneous localization, of discriminating two

points, and of recognizing the finer grades of temperature, called cool and warm."

These three forms of sensibility are transmitted from the periphery by means of the posterior roots into the spinal cord. Anatomically, the *posterior roots are supposed to contain five* different sets of fibers, each having a different termination.

The *first* goes to the nerve-cells of the anterior horns of the same side and is supposed to subserve reflex functions.

The *second* ends in the cells of Clarke's column, this consisting of a group of cells situated at the base of the posterior horns, running along the whole extent of the spinal cord, but principally from the first thoracic to the second lumbar. From these cells new fibers arise which run to the postero-lateral portion of the same side of the cord, forming the so-called direct cerebellar tract, lateral to the motor columns and behind the tract of Gowers. This tract maintains this position in the spinal cord and enters the cerebellum by means of the inferior cerebellar peduncle and is supposed to end in the superior vermis.

The *third* set of fibers ends in the nerve-cells of the posterior horns on the same side. From here new fibers arise, most of which cross over in the gray and white commissure, forming the so-called Gowers' tract, situated anterior to the direct cerebellar and outside of the lateral motor columns. These fibers ascend in the spinal cord and in the lateral portions of the medulla and pons, terminating in the lateral and ventral nuclei of the optic thalamus. From here a new set of fibers arise, these ending in the sensory cortex. Some of the fibers of Gowers' tract, however, are given off along their course and end in the superior vermis of the cerebellum and in collateral nerve-cells situated in the medulla. Gowers' tract also receives a number of fibers from the cells of the posterior horn on the same side.

Not all the fibers arising from the cells in the posterior horns ascend in Gowers' column. A number cross over in the white commissure, forming the so-called anterior tract, situated in the anterior portion of the cord in front of the anterior horns. Their course is not accurately known, but some of the fibers ultimately reach the nuclei of the optic thalamus, and from here a new system is given off to the sensory cortex.

The *fourth* system of fibers enters the posterior columns, forming the tracts of Gall and Burdach, and ascends in the spinal cord, terminating in the nucleus cuneatus and gracilis in the medulla oblongata. Every fiber after its entry in its course upward runs mesially, the fibers from the root above forcing it inward. Thus, in the column of Gall are transmitted the fibers from the posterior roots of the sacral, lumbar, and lower thoracic from the fifth to the twelfth, inclusive, while in the column of Burdach, which first becomes evident

in the fifth thoracic segment, are transmitted the fibers from all the cervical and the upper five thoracic roots. The fibers of the column of Gall terminate in the nucleus gracilis, and of the column of Burdach in the nucleus cuneatus. From these nuclei new fibers arise forming the internal arcuate fibers which decussate in the medulla and run upward, forming the so-called lemniscus or median fillet. These tracts occupy a position in the median line just back of the motor fibers in both the medulla and pons, and terminate in the lateral and ventral nuclei of the optic thalamus. From here new fibers arise which run through the posterior part of the posterior limb of the internal capsule, terminating in the sensory cortex.

The *fifth* arborize around the cells of the gray matter lateral to the horn. Their further course is not definitely known.

The three sensory neurons or systems.—We see, then, by tracing the course of the sensory fibers after their entry into the spinal cord, that there are three sets of neurons or systems, instead of two, as in the motor.

The *first* or peripheral has its center in the cells of the ganglia on the posterior roots. The fibers coming from these cells divide in a T-shaped manner, the external divisions going to the end-organs by means of the peripheral nerves, and the central part into the spinal cord, where they divide into descending and ascending branches. The descending branches are probably reflex in function, while the ascending transmit the different forms of sensibility, terminating in the nerve-cells in the gray matter of the same side of the spinal cord, or in the posterior column nuclei in the medulla oblongata.

The *second* neuron or system starts from these nerve-cells and ends in the median and lateral nuclei of the optic thalamus. It consists of the columns of Gowers, the anterior tracts, and median fillet.

The *third* neuron or system starts at the optic thalamus and ends in the cerebral sensory cortex.

It is important to keep the anatomic limits of the three neurons in mind, for the sensory disturbances resulting from lesions will in each instance differ. In the peripheral or first system the sensory symptoms will depend first of all upon the particular nerve or root diseased and its distributions, and alterations will occur in the three different forms of sensibility—the deep, protopathic, and epicritic. Just as soon, however, as these peripheral types of sensibility arrive in the spinal cord, they are readjusted, and touch, pain, and temperature sensations arriving, no matter from what system, are transmitted in the second neuron along definite tracts, and instead of having the three peripheral types of sensibility, there will be disturbance either of touch, pain, heat, or cold in its entirety.

In the third system or neuron there is again a readjustment, and instead of separate pathways, all the different forms of sensibility run together, and a lesion in any portion will cause disturbance either partially or totally of all forms.

The transmission of sensation to the cerebral cortex after its entry into the spinal cord.—Although we distinguish many different forms of sensibility, and while it is believed that each is transmitted along definite tracts in the spinal cord, it is probable that every type of sensation is transmitted along more than one tract.

Touch and muscle sense.—Sensation for touch probably ascends in the posterior columns or the columns of Gall and Burdach on the same side of the spinal cord to the respective nuclei in the medulla, and from there, after its decussation, by the median fillet to the optic thalamus, and then to the sensory cortex. It is also believed that touch sensation is transmitted by the heterolateral anterior columns. The important point is that destruction of one of these tracts will cause incomplete, while destruction of both causes complete, loss of touch sensation. Muscle sense which is transmitted in the peripheral nerves along different (muscular nerves) fibers from those of touch, ascends in the spinal cord with touch sensation, for nearly always loss of one will be associated with loss of the other. When considering loss of touch and muscle sensation, it must be remembered that most of the fibers concerned with these senses do not decussate in the spinal cord, and that unilateral lesions will cause a greater disturbance on the side of the lesion. This differs from the other (pain and temperature) forms of sensation because the fibers conducting them decussate in the spinal cord, and therefore unilateral lesions will cause the principal disturbance upon the opposite side. In testing for touch sensation it is best to employ cotton-wool on the non-hairy parts, or else the pressure of the examining finger will cause confusion by stimulating deep sensibility.

Pain and temperature sensations.—These are transmitted in the column of Gowers. While it is admitted that pain, heat, and cold are transmitted in these tracts, it is probable that separate fibers are concerned with each type, for it is possible to have disturbance of one without derangement of the other. In both the medulla and pons the fibers conducting pain and temperature sensations are apart from the other senses, but after their entry into the optic thalamus they are conducted with them through the posterior limb of the internal capsule to the sensory cortex.

In testing for pain and temperature sensation test-tubes with hot and cold water should be used. It is advisable in a given case to have these always of the same temperature.

Muscle sense.—By this term is meant every form of sensibility transmittted from the muscle, tendon, capsule, and joint concerned

with the movement of a part. As such it must be made up of the sense of position, of active and passive movement, of pressure, and of touch sensations. It is probable that this complex sense is transmitted with touch sensation on the same side of the cord chiefly in the posterior and somewhat in the lateral columns. The impulses in the posterior are concerned with the conscious appreciation of this sense, while that transmitted in the direct cerebellar tract to the cerebellum is concerned with unconscious coördination.

The ataxic gait always results from a disturbance of muscle sense, for example, in locomotor ataxia in which there is degeneration of the posterior roots and of the posterior columns, and differs from the asynergic gait of cerebellar disease in which there is no disturbance of sensation. In the ataxic the gait is always worse in the dark or when the eyes are shut. In the cerebellar patient this does not matter.

Sense of position.—The limbs should be absolutely relaxed and the eyes closed. When testing, for instance, the right upper limb it should be placed in a given posture and the patient should be asked to put the other limb in the same position, or to describe where it is or vice versa.

Sense of movement.—The test is similar to that of the sense of position, the patient being asked to describe the movement or have the opposing limb moved in accordance with the movement of the examiner, or vice versa.

Ataxia.—By this is meant an incoördinate movement. In some diseases, as in locomotor ataxia, when the posterior columns are degenerated, the incoördination is increased with the eyes shut. In testing the upper limbs the patient should be asked to put the finger to his nose or to put the fingers together. In testing the lower limbs it is best to place the patient on his back and have him move the heel to the knee, leg or toe. In testing for the whole body, the patient should be asked to stand up and put his heels and toes together. If the patient staggers and falls, it is sometimes called *Romberg's sign.* The ataxic gait may be due to a disease of the posterior columns of the spinal cord, as in locomotor ataxia, and differs from the asynergic gait of cerebellar disease.

In the *cortex* the sensory functions have been located entirely back of the central fissure, in the post-central, the posterior portion of the para-central convolution and the superior and inferior parietal convolutions. These areas on the right side of the brain control such functions on the left side of the body, and vice versa.

Sensation is again rearranged in the cortex into the so-called *primary and acquired sensations.* By primary sensations are meant the senses of pain, temperature, and touch. To this may be added the so-called bone sensation. These are first developed in the growing

child. Later on, the so-called acquired sensations or the senses of movement, position, localization, and pressure are attained.

The *primary sensations* are localized in the post-central convolution and the posterior portion of the para-central convolution. There is undoubtedly a subdivision of these centers similar to that for the motor centers in the pre-central convolution, the head center being below, the arm center above, and the leg center on top. Irritating lesions of the primary sensory centers cause pains upon the opposite side of the body, depending upon the point of irritation. These pains are usually described as burning sensations and begin in the part of the body corresponding to the point of irritation, and extend accordingly. These are analogous to Jacksonian convulsions, but they are by no means of as much value, for pains can be the result of many different lesions and their character is by no means distinct.

While a destruction of the whole of the post-central convolution will cause loss of sensation on the opposite side of the body, that is, hemianesthesia, yet this is of very infrequent occurrence, for permanent hemianesthesia due to cortical lesions is rarely seen.

It is probable that the cortical sensory centers are subdivided functionally in the same way that motor centers are, and that a lesion of a part of the primary sensory centers will cause disturbance of sensation in those parts which are in relation to a movement, just as a disturbance of a part of the motor centers gives impairment of movement. The difficulty in sensory delimitation, however, is evident, for if the definition of a movement is difficult, sensory delimitation is even more so. Therefore, in partial lesions of the post-central convolution there would be disturbance in limited portions of the limb or limbs of a movement-like character, and which as had been emphasized, are very difficult to outline.

The *so-called acquired sensations* or the senses of movement, position, localization, and pressure are located in the superior and inferior parietal convolution. These functions for the upper limb are in the inferior parietal convolution and part of the superior parietal convolution, and for the lower limb in the upper portion of the superior parietal convolution.

If these functions are normally developed every movement of a limb is performed with precision. If there is a disturbance of any or all of these functions the movements will be uncertain or, as it is called, *ataxic*. In parietal lobe lesions of the left side of the brain such disturbances are present in the right side of the body, and vice versa. Such an ataxia can be distinguished from that of locomotor ataxia in the fact that in the latter disease this symptom is present in the limbs on both sides.

To summarize, experience has shown that limited cortical lesions are hardly ever confined to either the post-central or parietal convolutions alone and that symptoms of such lesions not only produce disturbance of the so-called primary sensations, but also of muscle sense or the acquired sensations. Therefore, it can be taken for granted that cortical sensory lesions give disturbance of touch, pain and temperature, and impairment of muscle sense or of sense of movement, position, localization pressure and astereognosis in part or parts of the limbs on the opposite side.

The stereognostic function and astereognosis.—The ability to recognize an object placed in the hand is largely an intellectual function, for if all the forms of sensation are normal, an individual can tell the form, shape, weight, and size of an object, yet he can not name it unless the name of this object has been previously acquired. This ability to recognize objects by the hand is called the stereognostic sense. A destruction of this function is called astereognosis. The stereognostic senses are located in the superior parietal convolution on either side of the brain, and a destruction of either superior parietal lobe will cause astereognosis on the opposite side.

Astereognosis is tested for in the upper limb by the recognition of different objects placed in the hand. It can be tested for in the foot by having an object placed against the sole, for individuals should be able to recognize the size and dimension of an object. But this sensation is not developed as much as it is in the upper limbs.

SENSORY SPEECH CENTERS.

Just as the motor centers of right-handed individuals are located in the left side of the brain, so the sensory speech centers are similarly located in the left side of the brain in right-handed individuals. In left-handed persons both the motor and sensory speech centers are located in the right side. Inasmuch as most of us are right-handed, the center on the left side of the brain only will be discussed.

The sensory speech centers have been located in the middle portion of the second left temporal convolution. The centers for audition or hearing have been placed in the middle portion of the first left temporal convolution. The centers for audition, however, are on both sides of the brain, but the sensory speech center is only on one side of the brain.

A disturbance of the sensory speech center will produce sensory aphasia. What is meant by sensory aphasia? The answer to this lies in the factors which constitute sensory motor speech. It is therefore well first to discuss the method in which speech or language is acquired.

A child learns to speak chiefly through the hearing of sounds. The modern or phonetic method of teaching is diffrent from the old-fashioned method in which a child was taught first to spell a word, the sound of the word being acquired afterward. In the present method a child is shown, for example, an apple or the picture of an apple, the sound of the word is acquired, and it is not until afterward that the spelling of the word is taught. While this method is much quicker than the old, yet it has had the effect of making the modern young man or woman a much poorer speller.

Speech is acquired chiefly through sound or word hearing and through word seeing, the two senses being perhaps equally important. With word hearing and word seeing, word writing is associated; in other words, language is acquired through hearing, seeing, and through the associated motor and sensory functions which are necessary for the purpose of writing. Of course, in the blind, word seeing does not enter, but in such case the spelling and recollection of letters and words is acquired through the fingers or through the sensory functions.

It is manifest that a person who knows more than one language will have a greater development of the sensory speech centers, by this meaning a greater development of nerve cells. There is a distinct center for figures. Inasmuch, however, as figures are always associated with speech, sensory aphasia always entails a disturbance of figures. There may be disturbance or loss of sensory speech without impairment of the knowledge of the sound of music, which while it is located in the temporal convolution yet is distinct from it. It is apparent that in some individuals the musical center is much more developed than in others, for there are many persons who are tone-deaf. It is also manifest that the greater the musical knowledge the greater would be the development of the musical center in the temporal convolution.

If, therefore, a person should have a lesion of the sensory speech centers, what would the symptoms be? Such a person would be word-deaf, that is, he would not be able to understand the meaning of the sound of words, and it would be impossible for him to understand what some one else would say to him. Secondly, because of the fact that he had lost the memory of the sound of words, he could not express in words the meaning he wished to convey, because he could not formulate in his own mind what he desired to express by means of the sound of words. Such a person, however, could talk, because his motor apparatus would be intact, but his speech would not express his meaning. He would jumble up his words in a meaningless manner, this being called *jargon* or *paraphasia.*

Because of the fact that language is acquired by both hearing and seeing, such a person would be unable to appreciate the meaning of

letters and words, and would not be able to read a paper or would not be able to express his thoughts in writing because his memory for written words and letters would be lost. He would be able to write, however, because his motor writing apparatus would be intact, but his writing would not express what he wished to say. In other words, it would be a jargon, writing aphasia.

The *center for word seeing* has been located in a contiguous portion of the brain, in the adjoining angular gyrus, this convolution being around the end of the first temporal fissure in the left side of the brain in a right-handed individual, and vice versa. In this angular gyrus has been placed the center for the memory of word and letters images. A lesion of the angular gyrus will cause inability to write spontaneously because the memory of the meaning of written words and letters has been lost, and inability to read or to understand written words and letters would follow. Such a person, however, could write, or copy letters and words, but he would not know what he was copying. This is called *alexia.*

While, of course, there are isolated cases of lesion of the angular gyrus just as there are isolated cases of lesions of the second left temporal convolution, yet there is not a recorded case of pure word and letter blindness or of pure sensory aphasia without some involvement either of word hearing or word seeing, although theoretically such a condition may exist.

Unequal development of the word hearing and seeing centers is seen in both pathological and normal cases. For example, in a person who knows a number of languages a gradually destructive lesion will always affect the language which has been last acquired, the knowledge of the mother language persisting the longest. Again, in such persons it may be that the visual functions are affected last, or, on the other hand, they may be involved first.

Unequal development in the healthy person is very well shown among medical men. It is very rare to see a physician discuss the subject of a paper if that paper is not illustrated. It has seemed to me that visual development, at least in physicians, is much greater than the word hearing development. Of course, this depends largely upon one's education. I know of a very distinguished Philadelphia physician whose sight is considerably impaired and who has been read to for many years. This person has been trained to listen to such an extent that he can quote verbatim any reader's paper; whereas, on the other hand, another, a distinguished microscopist, never discusses a paper unless it is illustrated by charts or figures.

Of course, there are variations in the extent of sensory aphasia. In *complete sensory aphasia*, word hearing and seeing are entirely lost. In *incomplete* sensory aphasia the loss of word hearing is

comparable to loss of word seeing, the completeness of the aphasia, of course, depending upon the extent of the lesion.

If there is a lesion of Broca's convolution in addition to those of the temporal convolutions, there will be a *combined motor and sensory aphasia* in which there will be inability to understand the meaning of the sound of written words, besides inability to talk. Such a condition should be common in large shrapnel wounds of the head. There are, of course, variations in the degree of the aphasia, this depending upon the completeness of the lesion. In civil life such an aphasia is nearly always the result of the closing of the Sylvian artery on the left side.

The theories of motor and sensory aphasia which I have taught are those which have been generally accepted for many years and have stood the brunt of time. About five years ago Marié upset many of the older theories, claiming that there was no such thing as motor or sensory aphasia, but that both motor and sensory aphasia were only variations of one common type of aphasia, this depending upon an intellectual defect produced by a given lesion. There is no question that any large cortical or brain lesion must produce an intellectual defect. This is at once apparent when one considers the method of the acquiring of language. It is therefore taken for granted that there is a certain amount of intellectual defect in every aphasia, the degree depending first of all upon the extent of the lesion, and secondly upon the intelligence of the individual.

Visual cortical centers.—The visual centers have been located in the occipital lobe, the outer or lateral surface of which is separated from the adjoining parietal lobe by the parieto-occipital fissure. The mesial aspect of the occipital lobe is much more important than the lateral, vision being chiefly centered in the calcarine fissure, and the two adjoining lobes, located above and below, that is, the cuneus and the lingual lobes.

To appreciate the results of lesions of the occipital lobe it is necessary to know the fields of vision and their subdivisions. If the right eye is closed, whatever is seen with the left eye is called the left visual field. This is equally true of the right eye. The visual field has a definite boundary, being known as the form field. Color fields also have their definite boundary, but they are less than the form fields, the largest being the blue, then comes the red and green, these three colors being the usual ones tested for. Such tests can be made roughly by the hand, but usually should be done by the perimeter.

The *visual fields are divided* into temporal and nasal portions, the temporal being by far the larger. They are again divided into quadrants, this being done by a line drawn through the middle of each field of vision. If there should be loss of vision of the right temporal

and left nasal field of each eye, such loss of half-vision is called a right *hemianopsia.* Similarly a loss of vision in the left temporal and left nasal fields of each eye is called a left hemianopsia. A loss of vision in corresponding quadrants is called a *quadrant anopsia.* If there should be, for example, a loss of vision in the right upper quadrant of each eye it would be called right upper quadrant anopsia, etc. Not only is it necessary to test for the form and color fields, but when studying fields of vision it is necessary to observe whether or not the so-called *central* or *direct vision* is implicated, for it has its cortical representation.

Each occipital lobe supplies half-vision of each visual field. For example, a destruction of the right occipital lobe will cause loss of vision in the left half of each visual field, or a left hemianopsia. Similarly a destruction of the left occipital lobe will cause a loss of vision in the right temporal and left nasal fields, or a right hemianopsia.

The *cortical center* for central vision is probably represented either in the mesial or lateral surface of the posterior poles of the occipital lobes. *Macular representation* is unilateral and probably on the margins and the lateral surface of the occipital pole. In the upper lip of the calcarine fissure and the adjoining cuneal lobe is located the center for vision for the lower quadrant, and in the lower lip and the adjoining lingual lobe the center for vision for the upper quadrant. Therefore, a destruction of the right cuneus will give a left lower quadrant anopsia or a disturbance of vision in the lower quadrant of the left temporal and right nasal fields. Similarly a destruction of the right lingual lobe will cause a left upper quadrant anopsia.

It is possible to recognize a destructive from an irritating cortical lesion, for irritating lesions will cause so-called scintillating scotoma or flashes of light in the relating visual fields. For example, a right irritating lesion will cause flashes of light of different colors in the left visual fields, whereas a destructive lesion will cause an impairment of the form and color fields.

It is evident, therefore, that the visual fields are very important in cranial localization. Inasmuch, however, as disturbances in visual fields can occur from a lesion not only in the visual cortex but in the fibers coming from them, it is necessary to trace these fibers.

The temporal fields of vision are supplied by the fibers of the nasal retina, and the nasal fields are supplied by the temporal retina. The fibers from each temporal retina in their course backwards are to be found on the outer portion of the optic nerve, optic chiasm, and optic tract. The fibers from the nasal retina go to the inner portion of the optic nerve, then decussate in the optic chiasm, and are then found in the inner portion of the optic tracts.

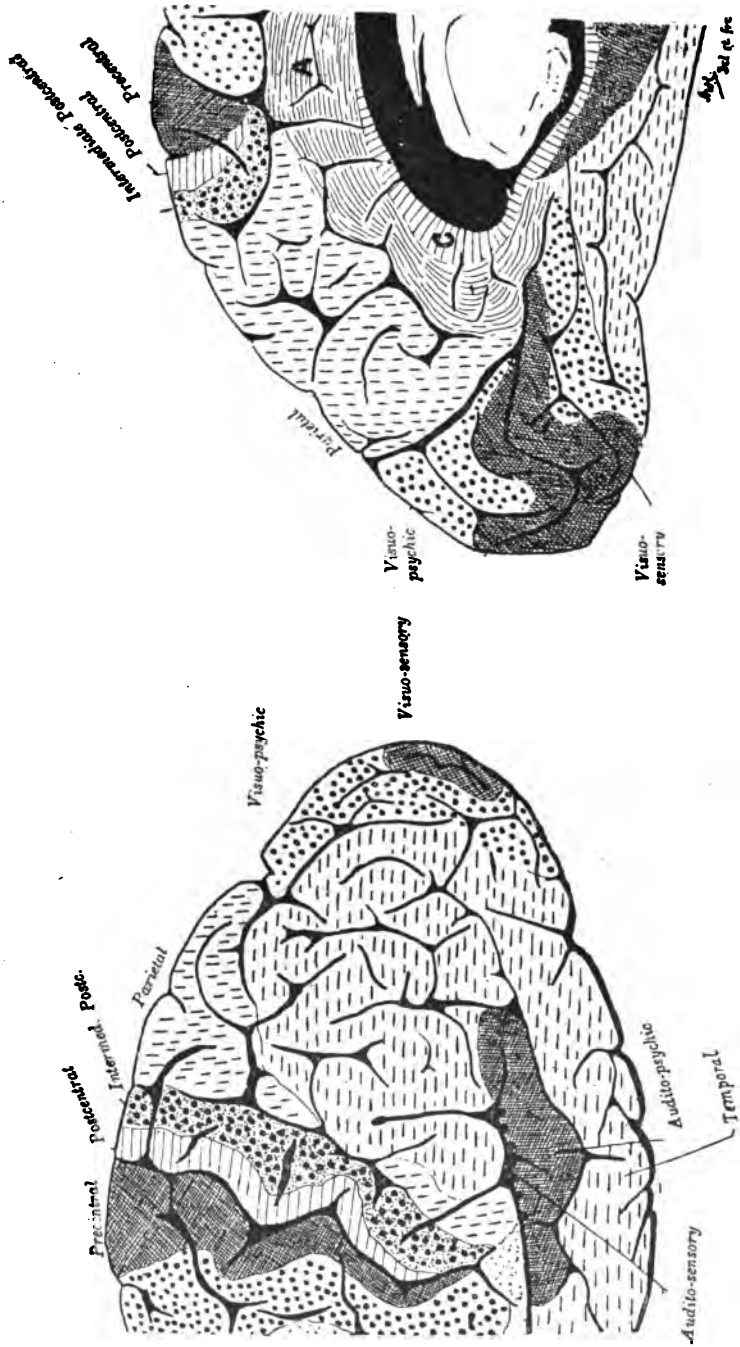

FIG. 15.—The distribution of the area striata (visuo-sensory) on the mesial and lateral surfaces of the hemisphere. (Campbell.)

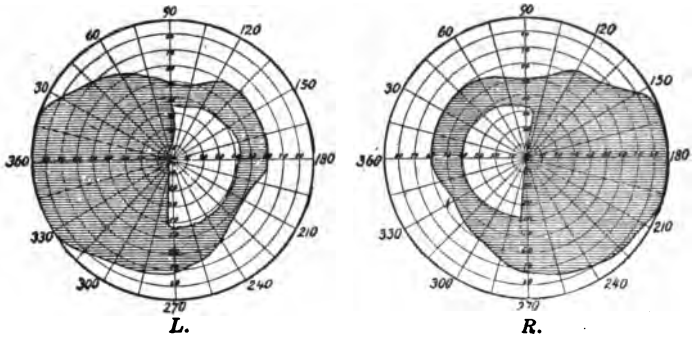

FIG. 16.—Bi-temporal hemianopsia. The shaded areas represent the portions of the fields which are dark, and it is evident that there are entire loss of both temporal fields and some contraction of the preserved fields (from a case of acromegaly).

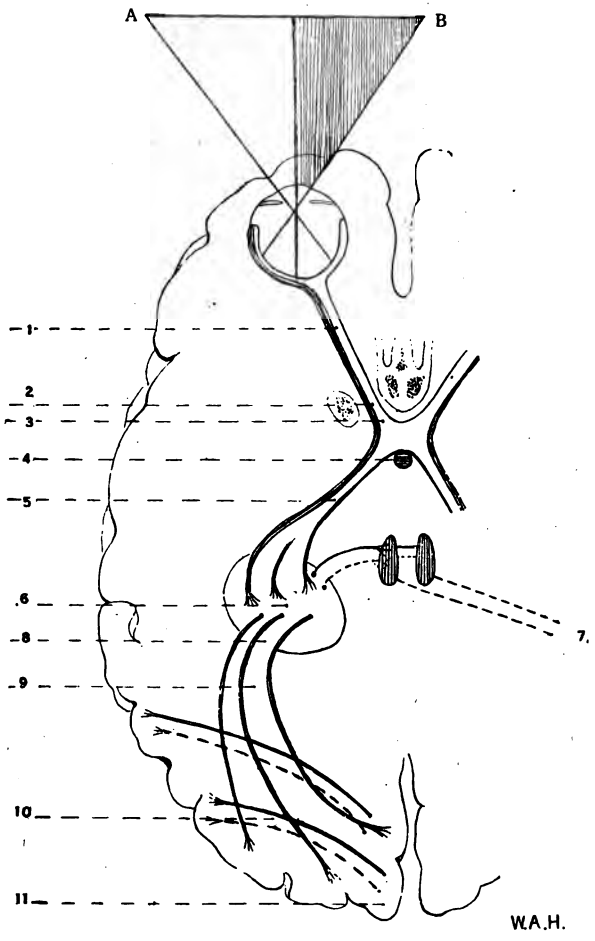

FIG. 17.—A diagram of the left visual pathway.

The dark outer portion of the optic tract and nerve represents the uncrossed fibers, which run to the temporal half of the retina and correspond to the shaded nasal half of the visual field B. The light inner portion of the tract and nerve represents the

fibers which cross in the chiasm and run to the nasal half of the retina and correspond to the unshaded temporal half of the visual field A.

1. *Optic nerve in the orbit.*—Pressure from hemorrhage, edema, or tumor causes various degrees of amblyopia and various contractions of the field.

2. *Optic nerve in the optic foramen.*—Pressure here from diffuse periostitis following ethmoiditis causes first a central scotoma and later complete blindness. Pressure from localized luetic periostitis causes first a peripheral defect in one portion of the field. Pressure here after fracture of the bone through the foramen causes very poor central vision and usually a lateral or vertical hemianopic defect in the field.

3. *The optic nerve just anterior to the chiasm.*—Pressure here from tumor of the frontal lobe causes first central scotoma and later blindness.

4. *The infundibulum of the pituitary body.*—Dilatation of the infundibulum or enlargement of the pituitary body compresses the crossing fibers in the chiasm and causes defects in the temporal half of the field of each eye.

5. *Left optic tract.*—Pressure here causes right homonymous hemianopsia with pallor of the optic discs.

6. *The external geniculate body and the thalamus* in which end the first visual neurons coming from the retina. Association fibers run to the nucleus of the third nerve. nerve.

7. Disturbance of these association fibers causes reflex iridoplegia (Argyll-Robertson pupil).

8. The beginning of the second visual neurons, which form the *optic radiation of Gratiolet.* A lesion here causes right homonymous hemianopsia with possible involvement of the nerves supplying the ocular muscles and also the auditory nerve. A lesion posterior to the internal capsule region may cause hemiplegia or hemianesthesia as well as hemianopsia, without pallor of the optic discs.

9. A lesion of the optic radiation here may cause right homonymous hemianopsia without other symptoms.

10. *The association tracts* running from the calcarine fissure to the angular gyrus. A lesion here will cause hemianopsia and, if on the left side, alexia or agraphia as well. Destruction of the angular gyri and neighboring structures in both hemispheres, even with intact optic radiations, will cause complete mind blindness.

11. *The pole of the occipital lobe,* the center for central vision. Destruction of the pole of each hemisphere causes a central scotoma in the field of each eye.

From the optic tract the fibers from both the temporal and nasal retina then go into the cells of the primary visual centers which are in the pulvinar or the posterior portion of the optic thalamus, the external geniculate body and the anterior corpora quadrigemina. From these primary optic centers a new set of fibers emanate and go to the occipital cortex by means of the extreme posterior portion of the posterior limb of the internal capsule, or the optic radiations.

If the course of these fibers is borne in mind the lesion causing any disturbance of the fields of vision can at once be recognized. For example, a destruction either of the occipital lobe, the optic radiations, or of the extreme posterior portion of the posterior limb of the internal capsule will cause hemianopsia upon the opposite side, for it is evident that there can not be a distinction in the visual symptoms from a lesion in any part of this central visual neuron whether the lesion is in the cortex or the fibers coming from the cortex, providing the cortical lesion is complete. Partial cortical lesion, however, may involve only the upper or lower quadrant, whereas lesions either in the optic radiations or in the posterior limb of the internal capsule will always cause hemianopsia of the opposite side.

Experience in this war has shown that the visual fields in the lower quadrants are nearly always involved as the result of isolated lesions of the occipital cortex. This is because lesions producing such disturbances are found in the cuneal lobes or in the upper part of the occipital convolutions. Lesions in the lingual lobe or the inferior portion of the occipital lobe are nearly always accompanied at the same time by disturbances in the cerebellum. Such injuries nearly always either result fatally or are in such a condition that visual delimitation is made difficult.

Bi-temporal hemianopsia is caused by pressure upon the crossing fibers in the optic chiasm and is usually due to an enlarged pituitary body or a dilated infundibulum compressing the chiasm from behind, below, or above. In the beginning, there is a relative loss of the

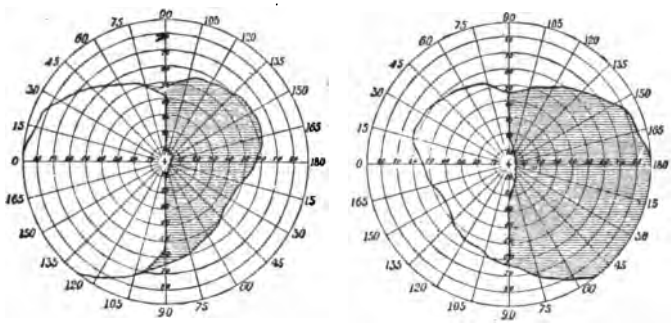

FIG. 18.—Left homonymous hemianopsia, from a case of gunshot wound, with suspected lesions of the right cuneus. The shading shows where vision was lost (from a patient under the care of the late Dr. S. Weir Mitchell in the Infirmary for Nervous Diseases).

temporal half of each field—that is, large test objects are recognized, but small white or colored test objects are not seen. Later, not even a light can be recognized in the temporal fields, and the upper or lower nasal quadrants also become involved. Finally, the fields may be lost entirely and complete blindness in both eyes result. A radiograph of the sella turcica will usually show enlargement or distortion of the sella.

Homonymous hemianopsia can be caused by disturbance of the visual fibers at any point between the chiasma and the cortex of the cuneus. The lesion occurs frequently in the internal capsule or in the cortex about the calcarine fissure, but is quite rare in the optic tract. In cases of tract lesion, the optic discs become pale, and accompanying basilar signs usually render the diagnosis possible. In lesions behind the optic thalamus the optic discs remain normal. The so-called hemianopic loss of pupillary reaction, when light is thrown upon the blind half of the retina, as a diagnostic sign of tract hemianopsia exists only in the minds of certain neurologists; the ophthalmologist does not find it.

When the lesion causing the hemianopsia involves the *corpora quadrigemina*, paralysis of ocular muscles and deafness are likely to be present. A lesion in the optic radiation, if near the thalamus and internal capsule, frequently is accompanied by hemiplegia of

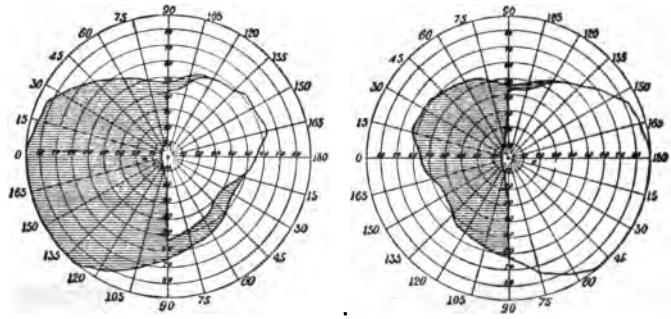

FIG. 19.—Right homonymous hemianopsia (from a patient under the care of the late Dr. Wharton Sinkler).

the same side and less frequently by hemianesthesia. Lesions further back, subcortical or cortical, may cause hemianopsia without other symptoms if the association tracts are not involved. These association tracts run from the visual perceptive center in the cal-

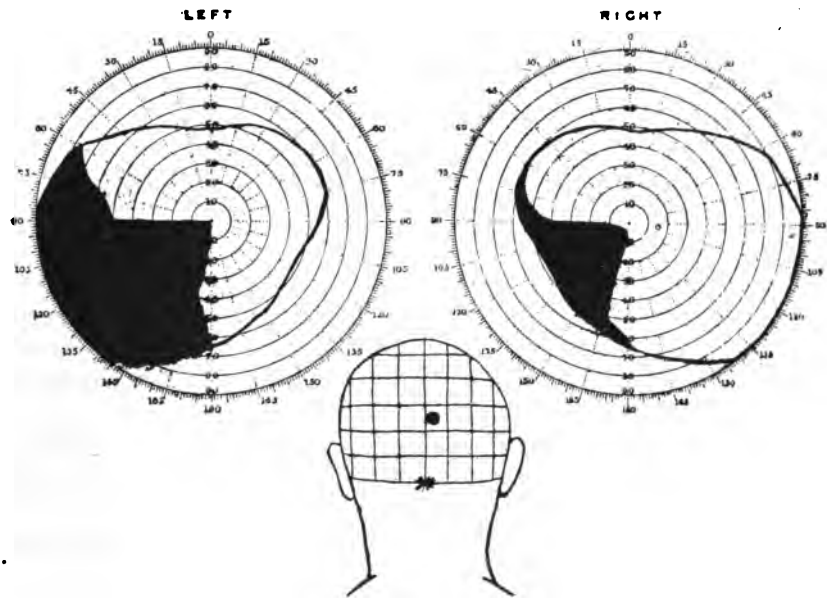

FIG. 20.

carine region on the mesial surface of the occipital lobe to the visuopsychic sphere which extends on the convexity of the lobe as far forward as the angular gyri. Destruction of these higher centers on both sides will lead to mind blindness. Destruction of the visual

fibers of the optic radiation, together with certain association tracts on the left side, will cause hemianopsia with alexia or agraphia.

It has been wrongly assumed by some that a so-called *hemiachromatopsis* indicates an involvement of a special color center. Hemianopsia, however, may range in degree from a slight relative to an absolute defect, and when the field is complete for large white objects but defective for one or more colors, it will be found that the field is equally defective for such small white objects as have a field equal to that for each color in the normal eye.

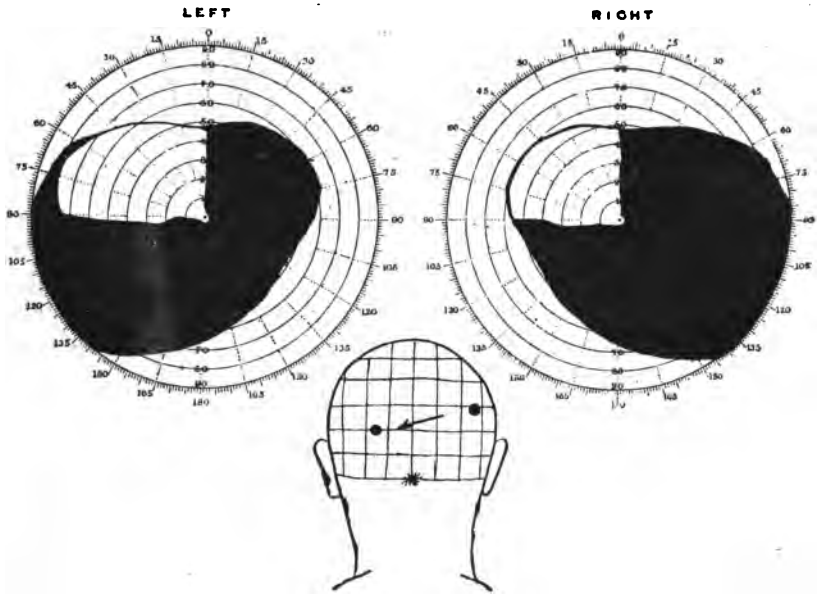

FIG. 21.

Gunshot injuries of the occipital lobe producing hemianopsia have been very frequent in recent wars, and the records of army surgeons have contributed not only to our knowledge of the clinical varieties of the hemianopic conditions but also in greater degree to our knowledge of retinal representation in the cortical visual area—the area striata. A paper by Gordon Holmes and W. T. Lister on "Disturbances of Vision from Cerebral Lesions" (*Brain*, 1916, p. 37), summarizing their records made in base hospitals in France, is a contribution of very great value. They group their cases of visual field defects into four categories:

I. *Quadrantic symmetrical defects* in the two fields, (*a*) one quadrant wanting, (*b*) two quadrants wanting, and (*c*) three quadrants wanting. The inferior quadrants were wanting much oftener than the superior, because a superior defect means a lesion in the inferior

portion of the area striata, which is likely to involve the cerebellum and be fatal.

II. *Central scotoma* in the field for each eye, usually symmetrical, due to injury of the tip of each occipital lobe.

III. *Homonymous hemianopsia* with central scotoma (the central scotoma representing a central or paracentral hemianopic defect on the side opposite to the complete hemianopsia) due to an oblique injury through the optic radiation on one side and the posterior tip of the occipital lobe on the other.

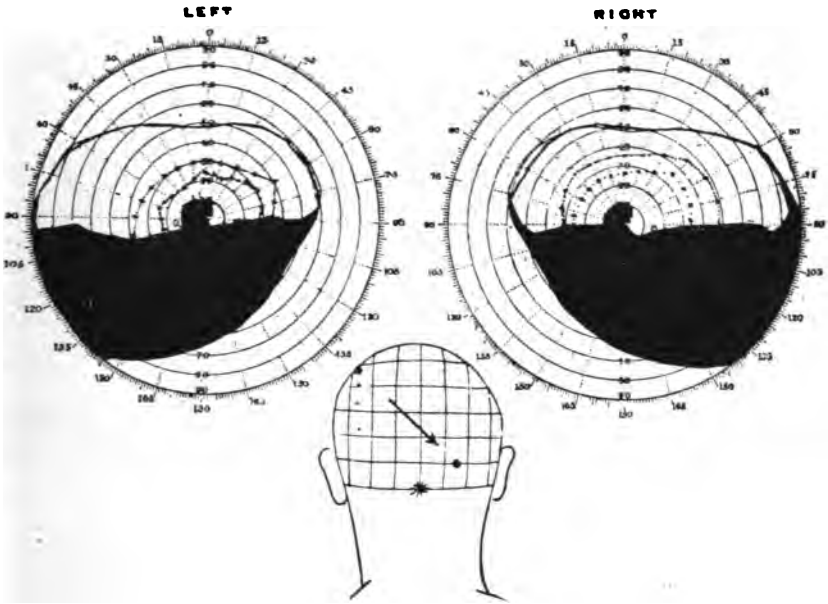

FIG. 22.

IV. *Paracentral symmetrical homonymous scotomata*, sparing central vision and being more extensive the further forward the injury.

These authors conclude, in brief, that each macula has a representation in one occipital lobe only, not in both, as had generally been believed; that the center for the macula lies in the posterior extremity of the area striata, probably on the margins and the lateral surface of the occipital poles; that the upper portion of the retina (and the lower portion of the field of vision) has its representation in the cortex above the calcarine fissure and the lower portion of the retina in the cortex below the calcarine fissure; and that the representation of the portion of the retina near the macula lies near the pole of the occipital lobe, while that of the periphery of the retina lies anteriorly.

It has long been recognized that when a right homonymous hemianopsia, for example, was later followed by a left homonymous hemi-

anopsia, due to a second plugging of an artery, a small central field was sometimes preserved. These authors explain such cases by call-

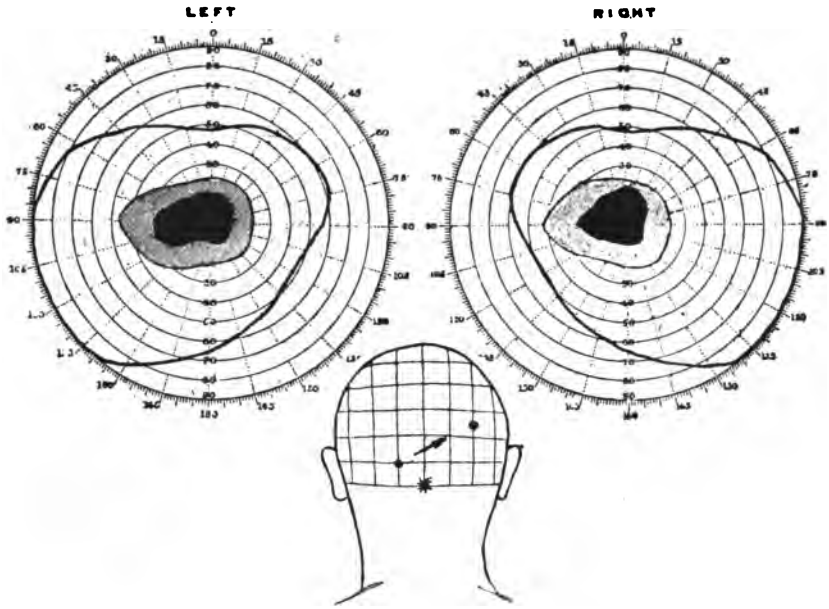

FIG. 23.

ing attention to the fact that the tip of the occipital lobe is often supplied by the posterior cerebral artery, while the rest of the area

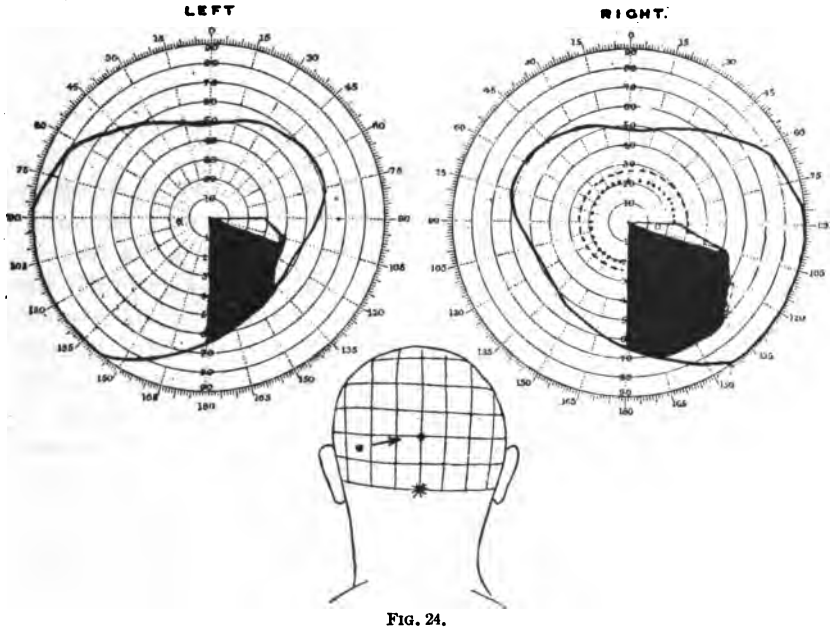

FIG. 24.

striata is supplied by the middle cerebral artery. Thus, plugging of the branches of each middle cerebral artery would produce bilateral homonymous hemianopsia, but might spare central vision in each eye, since the cortical centers for central vision might be supplied by the posterior cerebral arteries.

More recently Hine has placed on record a short series of cases of *occipital injuries characterized by recovery of portions of the fields.* The restoration occurred from the center toward the periphery and usually in the upper quadrant. The field for white returned before that for colors, and he appropriately advises the use of small colored test objects as well as white to avoid overlooking the field changes in these cases with rapidly changing and improving field phenomena. Riddoch has also made some important observations in studying the field phenomena associated with occipital injuries. He believes that dissociations of visual perceptions occur similar to disturbances in somatic sensibility. Further, that the elementary visual perceptions of light, movement, and of an object are dissociated in a manner similar to the dislocation of the sensory impressions of pain, touch, and temperature.

In cases with recovering fields, appreciation of movement and recognition of an object are always dissociated. The former is first recognized, begins in the periphery and can be charted. Later objects can be recognized, but in a smaller field. The absence of this dissociation after the lapse of a few months suggests an unfavorable prognosis for the return of vision.

Frontal lobe functions.—The frontal lobes constitute that part of the brain anterior to the pre-central convolution and are subdivided in the outer portion of the brain into four convolutions. For many years the centers for intelligence, reason, and memory have been placed in the frontal lobes, and it has been supposed that the left frontal lobe has more importance in this connection than the right. In recent years, however, considerable doubt has been thrown upon this theory, and the opinion is now widely held that intelligence is not limited to the frontal lobes, but that every part of the brain is concerned with it.

Take, for example, the question of language which we have previously discussed. We have learned that language is dependent upon the development of word hearing, word seeing, and word writing, and as more and more words are acquired there is correspondingly greater intellectual development. It is at once apparent, therefore, that language is not dependent upon one component, but that it is made up of a great many distinct factors, that while each one of these factors, such as word hearing, seeing, and writing, have their own cortical localization, yet they must act in harmony, and that

while a disturbance, for example, of the visual centers in the occipital lobe would give preponderant intellectual defect in seeing, and while similarly a disturbance of the word-hearing center in the temporal convolution would give preponderant defect in word-hearing, yet in both of these and similar instances which could be given there is a general defect in intelligence.

This point of view is not held by all neurologists. Charles Phelps has written of the localization of the mental faculties in the left pre-frontal lobe, but close analysis of his work and that of many others who have written similarly, discloses many errors. We could quote many examples of lesions in the frontal lobe in which no diagnosis was possible until an extension of the lesion into the pre-central convolution with the attending motor phenomena indicated the diagosis. A very good example of mistaken diagnosis is the following case of a physician of 70 who retired from practice at the age of 55. His son, also a physician, noted that his father began to go about the country calling on imaginary patients who had long been dead. He would hitch up his horse and buggy and make daily rounds and would constantly talk about the professional troubles that he had. His wife noticed that his memory began to fail and that he was slothful in his eating. We diagnosed a senile sclerosis, and it was not until he suddenly began to develop Jacksonian convulsions followed by a hemiplegic attack that the suspicion grew that he had a frontal tumor on the left side. Operation and subsequent autopsy confirmed this diagnosis.

It might be argued that the mental symptoms present in this case were the result of the frontal lobe lesion, yet the fact remains that these were not sufficiently distinctive to make a correct diagnosis. The conclusion, therefore, must be reached that frontal lesions do not produce distinctive symptoms and that their presence is made apparent only when the adjoining pre-central convolution is affected, with the accompanying motor phenomena.

SUB-CORTICAL LOCALIZATION.

The outer portion of the brain is called the cortex. The cortex, which is about an inch and a half in thickness, consists of groups of nerve cells. From these cells fibers originate which go to the various portions of the brain and spinal cord. There are also nerve fibers which end in the nerve cells of the cortex. There are three sorts of fiber tracts, (1) the *commisural system*, whose function is to correlate or associate one side of the brain with the other; (2) the *association system*, which connects different parts of the brain with each other, and, lastly, the so-called *projection system*, whose fibers chiefly go from the cortex of the brain into the spinal cord.

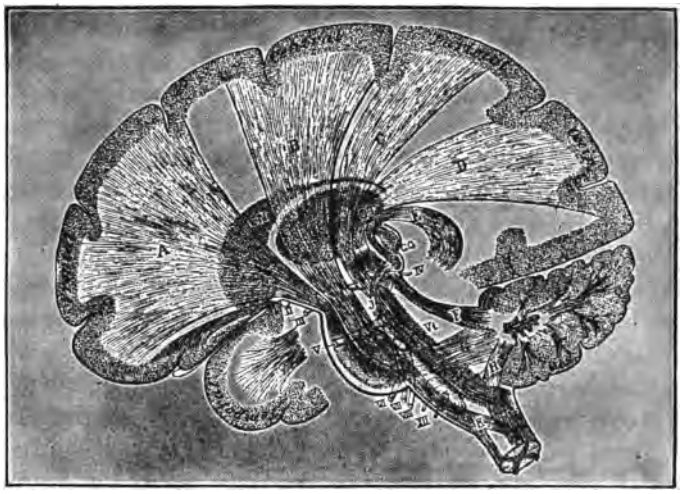

FIG. 25.—The projection tracts joining the cortex with lower nerve centers. Sagittal section, showing the arrangements of tracts in the internal capsule. The numerals refer to the cranial nerves (Potts).

A, tract from the frontal lobe to the pons, thence to the cerebellar hemisphere of the opposite side. *B*, motor tracts from the central convolutions to the facial nucleus in the pons and to the spinal cord; its decussation is indicated at *K*. *C*, sensory tract from posterior columns of the cord, through the posterior part of the medulla, pons, crus, and capsule to the parietal lobe. *D*, visual tract from the optic thalamus (OT) to the occipital lobe. *E*, auditory tract from the internal geniculate body (to which a tract passes, from the *VIII N* nucleus, *J*) to the temporal lobe. *F*, superior cerebellar peduncle. *G*, middle cerebellar peduncle. *H*, inferior cerebellar peduncle. *CN*, caudate nucleus. *CQ*, corpora quadrigemina. *Vt*, fourth ventricle.

FIG. 26.—The association fibers (Potts).

A, between adjacent convolutions. *B*, between frontal and occipital areas. *C*, between frontal and temporal areas, cingulum. *D*, between frontal and temporal areas, fasciculus uncinatus. *E*, between occipital and temporal areas, fasciculus longitudinalis inferior. *CN*, caudate nucleus. *OT*, optic thalamus.

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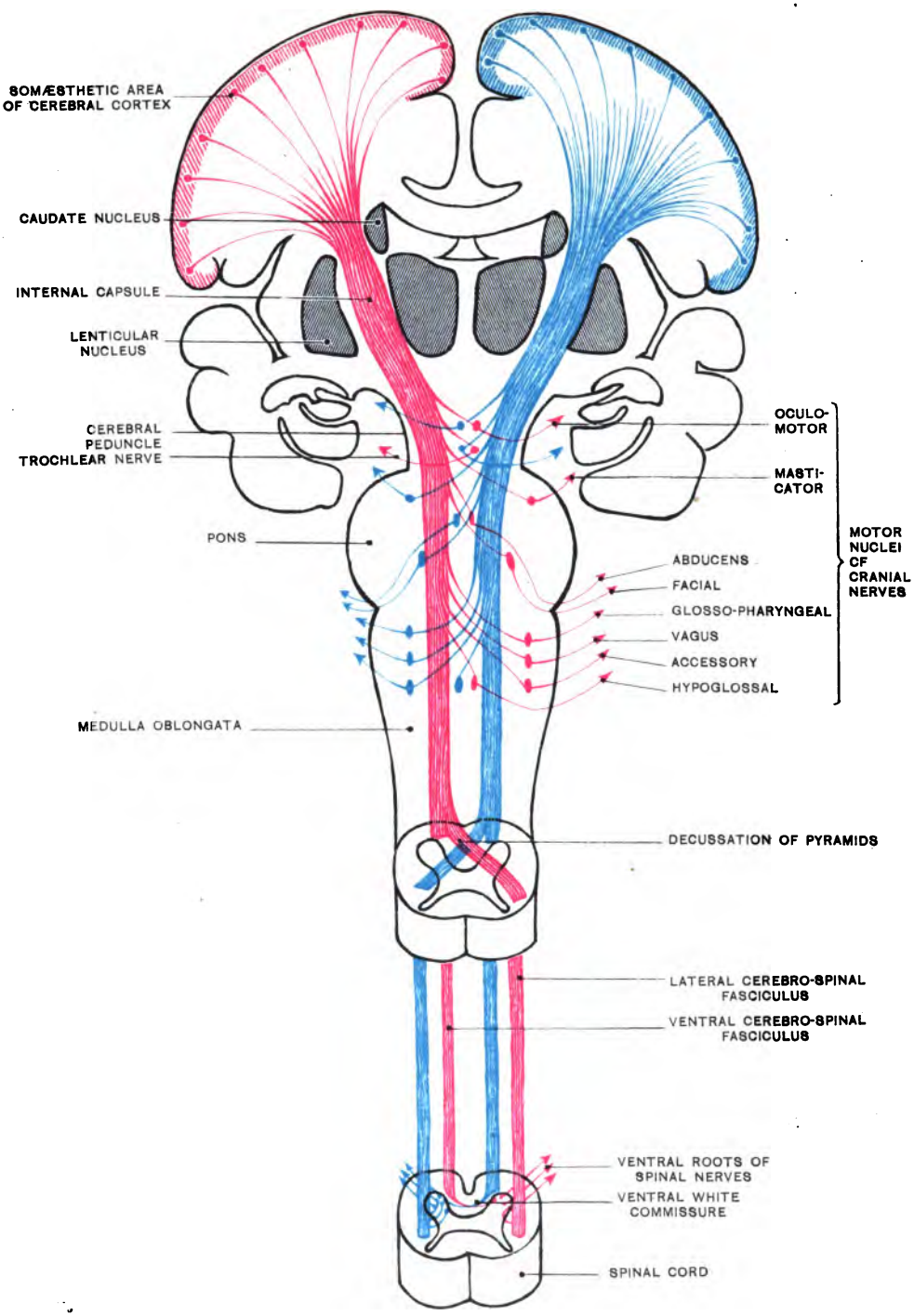
The *association system* plays a very important part in the normal functioning of the brain structure. It has already been pointed out in discussing language and intelligence that every part of the brain is concerned in the function. That being the case, every portion of the brain has to be connected with every other part. This is accomplished by the association tracts of which there are two kinds, the short and long. The short connect adjoining areas, whereas the longer tracts connect the more distant parts. The corpus callosum connects the lateral hemispheres with each other. It is at once apparent that if any part of the brain is injured not only will there be a destruction of the involved cells but there must be a secondary degeneration of the fibers leading from the point of injury. Such a degeneration must produce impairment of function in the part of the brain with which a tract is connected. Therefore it is a rule in all brain injuries that every traumatism will produce two sorts of disturbance of function. One, an impairment of that function which is directly injured, and, secondly, impairment of the function of the distant part of the brain with which the injured area is connected. This has been termed diaschisis.

Keeping in mind, therefore, the thinness of the cortex and the fact that the white matter underneath the cortex is composed chiefly of association fiber tracts, any lesion which involves more than the cortex can not have purely cortical or distinctly focal symptoms. The *symptoms of a sub-cortical lesion*, therefore, will be more diffuse and greater in number and will of course depend upon the extent of such involvement and the point of injury. A sub-cortical lesion of the motor area will give chiefly motor symptoms, the parietal lobe largely sensory symptoms, etc., but these symptoms will not be clear-cut. It is also apparent that lesions of the brain are hardly ever purely cortical. Pure cortical lesions nearly always result from direct trauma or are secondary to disease of the meninges.

The *projection system* (see Fig. 27) is composed chiefly of those fibers which go to the spinal cord. These fibers from every part of each lateral hemisphere, the frontal, pre-central, post-central, parietal and occipital lobes, converge in a central point and form what is known as the *internal capsule*, which is nothing more than a pathway for fibers. The internal capsule consists of an anterior limb, knee, and posterior limb. The anterior limb is between the caudate nucleus and the lenticular nucleus, and the posterior limb between the optic thalamus and the lenticular nucleus. The anterior limb transmits the fibers which come from the frontal lobes. The knee and anterior third transmit the the motor fibers, that is, the fibers coming from the pre-central and anterior portion of the paracentral convolution, the middle third of the posterior limb transmits the sensory fibers from the post-central and parietal lobes, and the



From Morris' Human Anatomy.

Fig. 27.

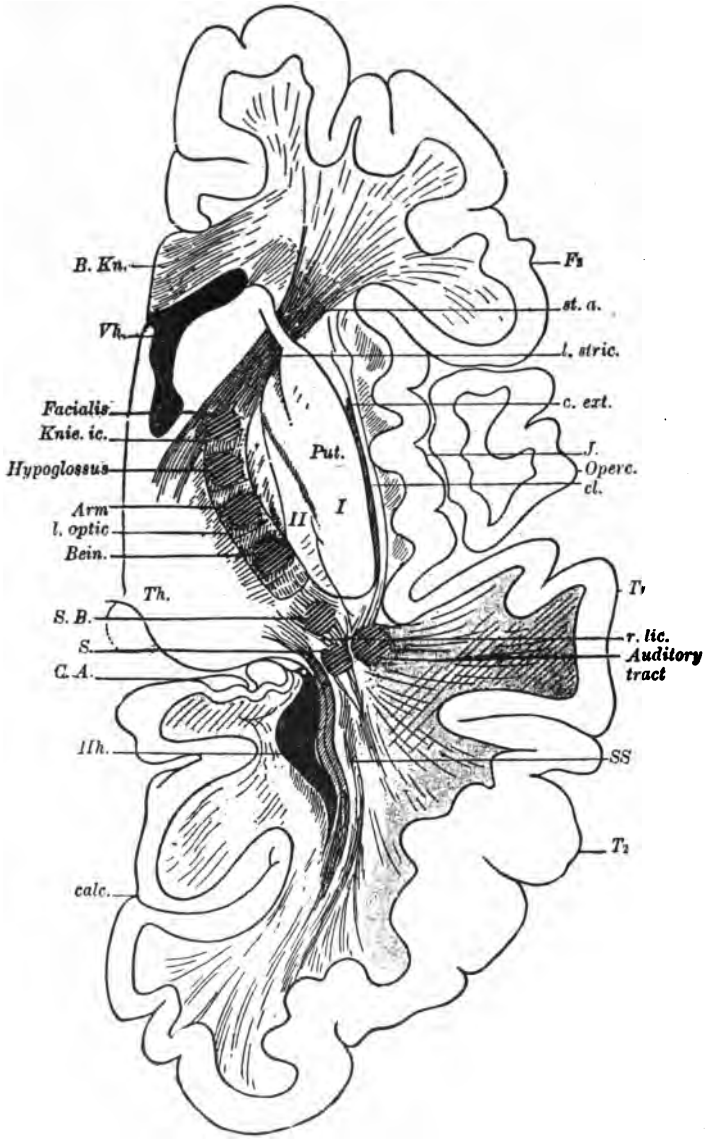


FIG. 28.—Horizontal section through the right hemisphere. (M. Allen Starr, after von Monakow.)

B. Kn, knee of corpus callosum. *Vh*, anterior horn of lateral ventricle. *F_2*, inferior part of third frontal convolution. *l. stric*, lenticulo-striate division of internal capsule. *Knie, ic.*, knee of internal capsule. *l. optic*, lenticulo-optic division of internal capsule. *Th*, optic thalamus. *J*, island of Reil. *cl*, claustrum. *T_1*, first temporal convolution. *Operc*, operculum. *r. lic*, retrolenticular region of internal capsule. *C. A.*, hippocampus major. *calc*, calcarine fissure. *Hh*, posterior horn of lateral ventricle. *SS*, optic radiation of Gratiolet. *T_2*, second temporal convolution. *S. B.*, sensory fibers. *Facialis*, position in capsule of motor tract to the face. *Hypoglossus*, position of tract to the tongue. *Arm*, position of tract to the arm. *S*, visual tract. *Bein*, position of tract to the leg. *A*, auditory tract.

posterior third of the posterior limb transmits the visual fibers from the occipital lobes.

There is a definite arrangement for the motor fibers in the posterior limb of the internal capsule. The knee transmits the motor fibers from the so-called head center, this consisting of those motor fibers which go to the motor cranial nuclei, that is, the nuclei of the third, fourth, motor portion of the fifth, sixth, seventh, ninth, tenth, eleventh, and twelfth nerves. Adjoining these cranial nerve fibers are the arm fibers and then those of the leg. It is probable that a similar arrangement occurs for the sensory fibers, but this has not been definitely established. There is also no doubt a definite arrangement for the visual fibers, but this too is not definitely known.

A *lesion*, therefore, *of the posterior limb* of the internal capsule, which is about an inch in length, will give hemiplegia, hemianesthesia, and hemianopsia on the opposite side. It is the only location in the brain in which any such small lesion can cause these definite symptoms. It would be impossible for a cortical lesion to produce hemiplegia, hemianasthesia, and hemianopsia, for it would involve two-thirds of the brain, and undoubtedly such a large injury would produce immediate death.

Cerebral centers for bilateral acting functions, such as laughing, crying, eating, and swallowing.—While it is acknowledged that in the cortex are localized the centers for every motion, sensation, and special act, it must be remembered that this is only in so far as simple acts, like lifting a finger or moving a limb are concerned. Where, for instance, it is necessary to perform a complex act, such as talking, laughing, crying, eating, and swallowing, there must be some one place or center which coordinates the different functions which such an act must constitute. The speech centers, probably because of their importance, are largely localized in the cortex, but they also have representation in the sub-cortex. As to where the centers for laughing, crying, eating, and swallowing are, we are not certain, but we believe that they must be localized in some of the ganglia in the sub-cortex, among these, the optic thalamus and the lenticular and caudate nucleus probably playing the most important rôle.

The optic thalamus.—The thalamus is anatomically divided into two halves by the lamina interna, its internal portion consisting of the anterior and median nucleus, and its external of the lateral nucleus, which is divided into anterior, middle, and posterior thirds, and dorsal and ventral halves. It also contains the center, median and arcuate nuclei, and ventral nucleus, which are in the basal part of the thalamus. The pulvinar forms the posterior part. It is known

'he continuation of the fibers of the fillet end in the anterior part of the lateral nucleus, while the continuation of the

fibers of the superior cerebellar peduncle end in the posterior part of the lateral nucleus.

The functions of the thalamus are fairly well known. Inasmuch as most of the ascending sensory fibers are re-grouped in this organ, lesions of the dorsal and lateral portions give fairly well-defined sensory symptoms. Involvement of the termination of the superior cerebellar peduncle will produce certain cerebellar symptoms which

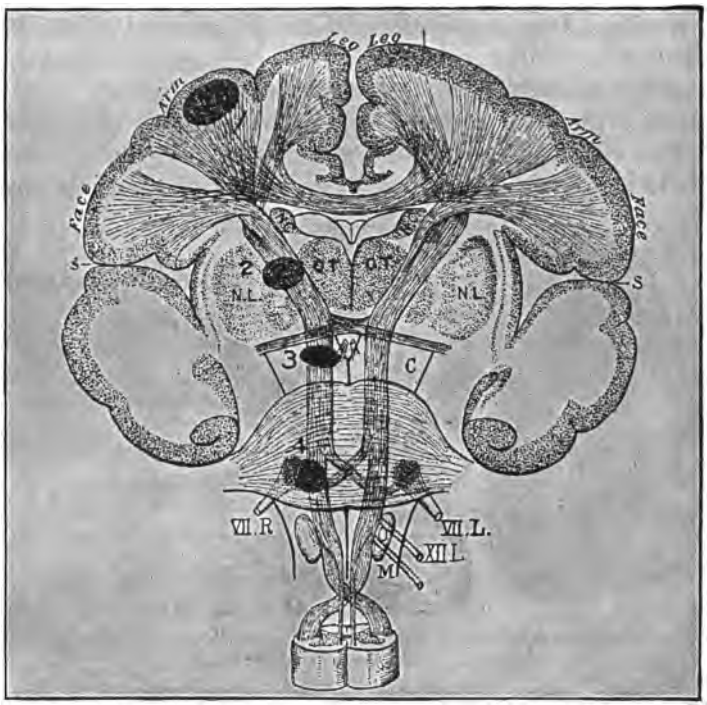


FIG. 29.—Scheme of the motor tract to show the effect of the lesions at different positions. (Starr.)

1, cortical or subcortical lesion, causing monoplegia of left arms. 2, capsular lesion, causing left hemiplegia. 3, crus lesion, causing left hemiplegia and right third nerve palsy. 4, pons lesion, causing alternating paralysis of right face and left arm and leg. *S*, sylvian fissure. *O. T.*, optic thalamus. *M. C.*, caudate nucleus. *N. L.*, lenticular nucleus. *VII*, facial nerve. *C*, crus. *M*, medulla.

are clinically manifested as irregular tremors and twitchings, whereas lesions of the pulvinar will cause severe visual disturbances.

A definite *thalmic syndrome* has been established. These symptoms may be either complete or incomplete, depending upon the extent of the lesion, and are manifested on the side opposite the lesion. These are:

1. Persistent loss of sensation of one-half the body and face. Deep sensibility, including deep pressure and postural sense, is much more implicated than touch, pain, and temperature.

2. Impairment or loss of muscle sense on one side, causing both hemiataxia and astereognosis.

3. Pains more or less persistent, coming on in paroxysms on the affected side.

4. Slight hemiplegia on the affected side because of the implication of the adjoining internal capsule. The hemiparesis usually is not marked and has a tendency to clear up.

5. Irregular movements on the affected side of an indefinite character, resulting from implication of the termination of the fibres of the superior cerebellar peduncle.

There has recently been described a curious sensory phenomenon consisting of an over-response to affective stimuli on the involved side. This consists in a response to stimulation which is more than what is to be expected and is of more or less disagreeable character.

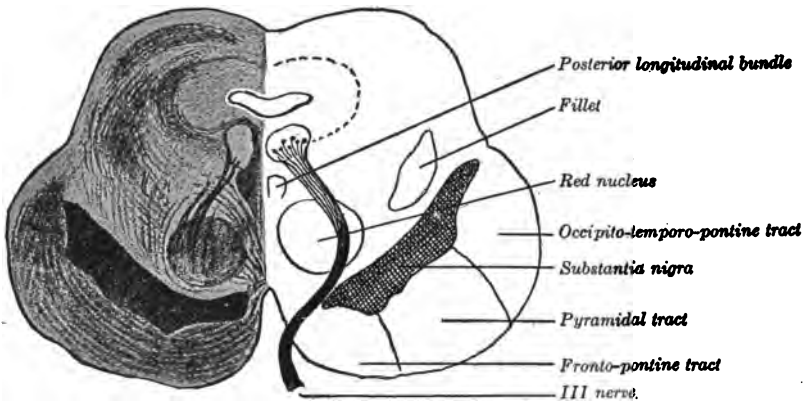


FIG. 30.—Section through cerebral peduncles and superior corpora quadrigemina (exit of oculomotor nerve).

Caudate and lenticular nucleus.—Nothing definite is known of the functions of these structures. Bilateral lesions of the lenticular nucleus sometimes cause involuntary laughing and crying and because of the recent work of Marié the lenticular nucleus has been considered a part of the zone of speech.

Lesions of the crus, pons, and medulla—The crus or cerebral peduncles.—The cerebral peduncles are practically the continuations of the posterior limbs of the internal capsule and transmit the fibers for motion and sensation, thus connecting the brain proper with the brain stem.

The nucleus of every cranial nerve, from the third to the twelfth, inclusive, receives its innervation from the opposite cortical center. The first and second cranial nerves do not enter in this, as they are really parts of the brain. The nucleus of the third or the oculomotor nerve is situated in the posterior portion of the crus, and its fibers have their exit at the foot of the cerebral peduncles.

A unilateral lesion, therefore, of the *cerebral peduncle* would always give *oculomotor palsy on the same side*, and *paralysis of the arm and leg only on the other side of the body.*

The pons.—In the pons are located the nuclei of the fifth, sixth, seventh, and partially of the eighth cranial nerves, the exits of these nerves corresponding in order. In a *unilateral lesion* of the upper part of the pons there will be *paralysis of the fifth nerve on the same side, with hemiplegia of the opposite side.* In a *lesion limited to the lower portion of the pons, there will result facial palsy on the same side and paralysis of the arm and leg only on the other side.*

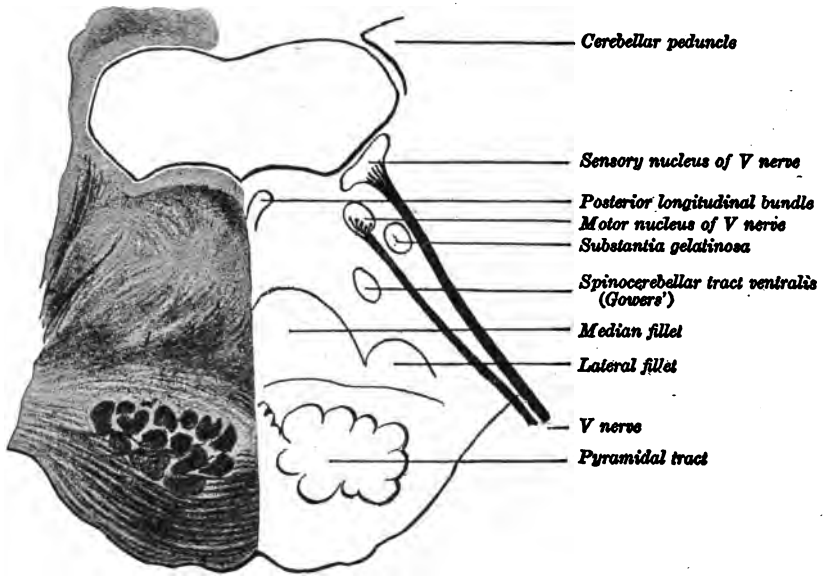


FIG. 31.—Section through pons at level of exit of fifth nerve.

In discussing the symptoms of lesions in the crus and pons, it has been assumed that these are confined to the anterior portions of these structures. Should the lesion, however, be more extensive, there would necessarily have to be involvement of the sensory fibers, which are located directly back of the motor, and there would result, in addition, sensory symptoms on the other side.

Paralysis of associated ocular movement.—Should, however, the lesions involve the median portions of the crus and pons, there would be paralysis of associated ocular movement. This is rather difficult to understand unless it is remembered that it is impossible to move one eye without the other, and therefore every movement of the eyeballs must be an associated movement. When we look to the right, we use not only the external rectus muscle on the right side, but also the left internal rectus, i. e., we are receiving innervation from the nuclei

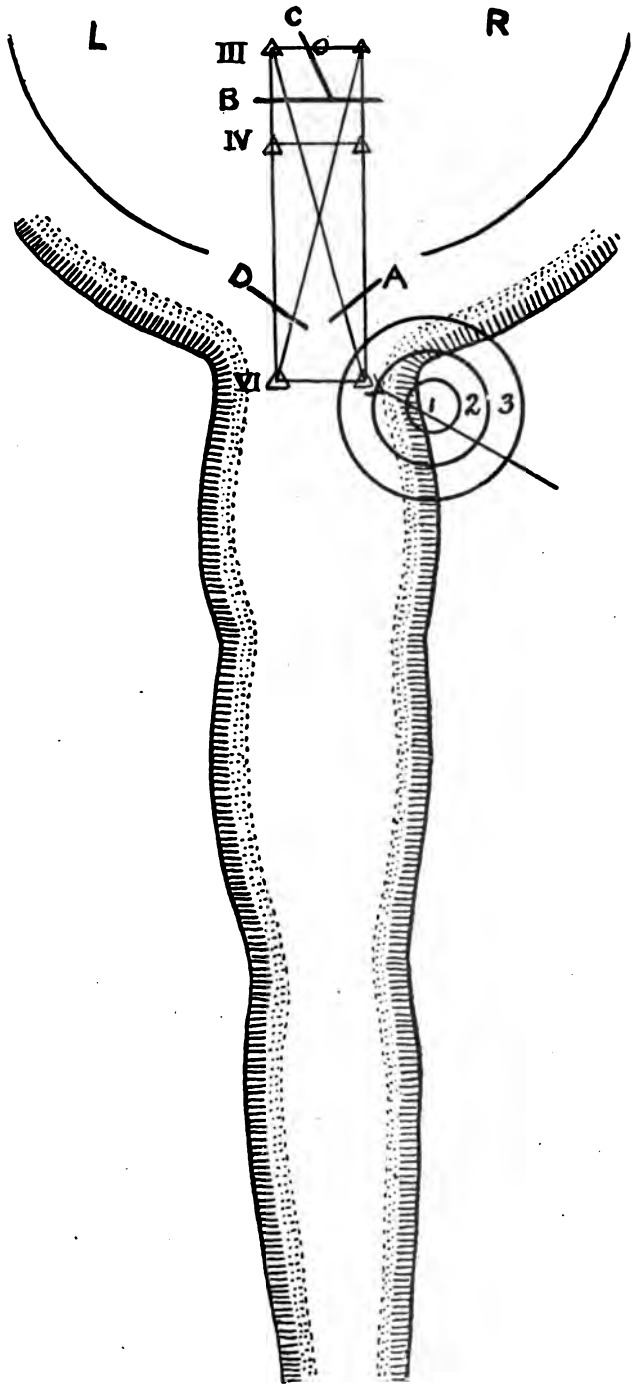


FIG. 32.—Posterior longitudinal bundle.

...sion A.—Paralysis of associated ocular movements to the right. Lesion B.—Inability to look down-
Lesion C.—Inability to look upward. Lesion D.—Paralysis of associated ocular movements to the
Lesion 1.—Motor palsy; sixth nerve involvement. Lesion 2.—Motor palsy; sixth nerve palsy; sensory
Lesion 3.—Motor palsy; sensory palsy; paralysis of associated ocular movements to the right.
cranial nerve palsies. (Weisenburg.)

of the sixth and third cranial nerves. To make this possible there must be a connection between these nuclei, and this is affected by the posterior longitudinal bundle, which is located in the posterior and median portions of the crus and pons.

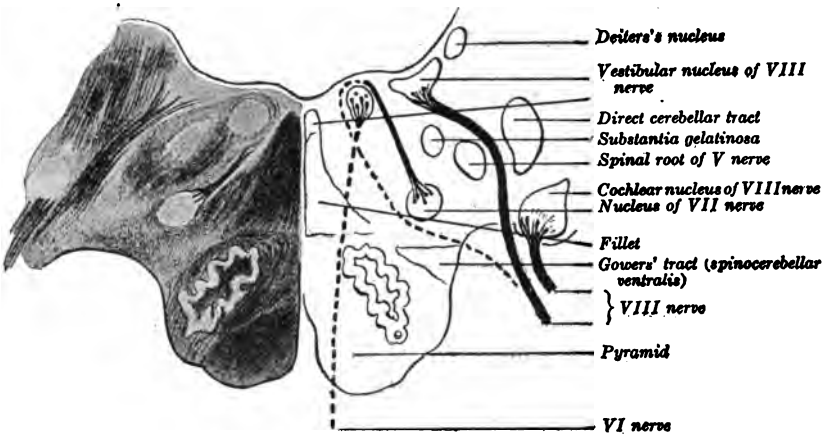


FIG. 33.—Section through lower pons, showing origins of sixth, seventh, and eighth nerves.

In looking downward we use not only the muscles which are innervated by the third, but also those which are innervated by the fourth cranial nerves. In looking upward we use only the muscles which receive innervation from both oculomotor nuclei. We see, then,

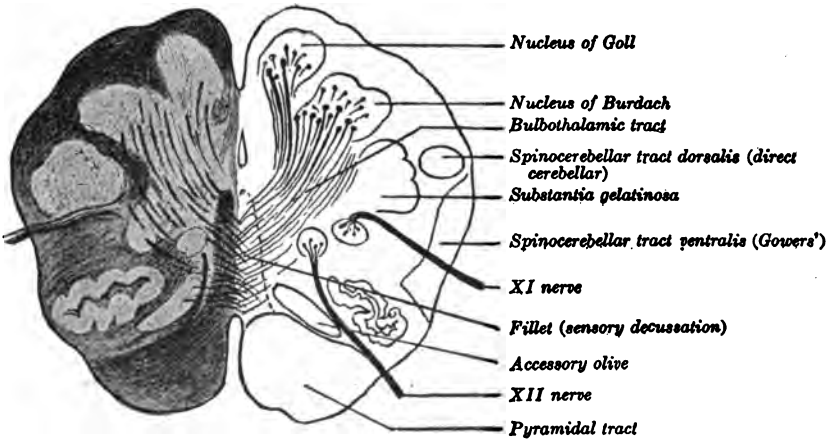


FIG. 34.—Section through medulla, level of hypoglossal roots.

that there must be a similar connection between the oculomotor nuclei and between them and the nuclei of the fourth nerves.

Should there be a lesion, for instance, in the lower part of the right side of the pons, cutting off the posterior longitudinal bundle, there will be inability to look to the right, and a similar lesion on the

left side will cause inability to look to the left. A lesion cutting off both bundles will cause inability to look to the right or left, but the ability to look upward and downward will be retained.

In a lesion of the upper portion of the pons which cuts off the connection between the third and fourth nuclei there will be paralysis of associated movement downward. A lesion still higher up will cause failure of upward movement.

Lesions of the anterior and posterior corpora quadrigemina.—The anterior corpora quadrigemina, in association with the pulvinar of the optic thalamus and the external geniculate body, is one of the so-called primary optic centers, and lesion of it should cause disturbance of half vision upon the opposite side. The corpora quadrigemini are so close together that nearly always both are involved. As a matter of fact, we know little of their functions, although it is presumed that the anterior are concerned with vision and the posterior transmit the central fibers of hearing. Nearly always lesions of this part are extensions of tumors, either from the aqueduct of Sylvius or more commonly from the pons, peduncle, or cerebellum. Because of the proximity of the third and fourth nuclei they will be involved early and bilaterally, and as the tumor grows the sensory and then the motor fibers become affected. Nearly always the fifth nerve is either unilaterally or bilaterally diseased.

Partial or total lesions of the medulla oblongata.—Isolated lesions are unusual. As a rule, tumors occupying this part are extensions from the pons or cerebellum. Hemorrhages are rare, and nearly always cause sudden death because of involvement of the ninth and tenth nerves. Thrombosis or embolism, especially of the inferior cerebellar artery, is not at all unusual, and is especially prone to occur on the left side. The symptoms in such a case will be unilateral disease of the eighth, ninth, tenth, eleventh, and twelfth nerves, with involvement of the motor and sensory fibers innervating the opposing arm and leg.

THE CEREBELLUM.

The *cerebellum* is situated in the back part of the skull in the inferior occipital fossa underneath the occipital lobes, from which it is separated by the tentorium cerebelli. It is composed of two lateral lobes and the middle portion called the vermis. The lateral portions as well as the vermis are divided into distinct lobules. Between and underneath the lateral hemispheres and the inferior portion of the vermis are found the pons, the medulla and its continuation, the spinal cord.

The outer portion of the cortex of the cerebellum is composed of large cells, known as the Purkinje cells. Underneath the cortex is a mass of white substance composed of myelinized nerve fibers. In

each lateral hemisphere is a group of nuclei, the largest of which is the dentate nucleus, and three smaller known as the nucleus fastigii, nucleus globosus, and nucleus emboliformis. These nuclei and the cortex are intimately associated by commissural and association fibers.

The cerebellum has three pairs of *peduncles* which connect it with the other nervous structures. The superior cerebellar peduncle connects the lateral lobes with the cerebral hemispheres. The fibers of the middle cerebellar peduncle come from the pons, and the fibers of the inferior cerebellar peduncle connect the cerebellum with the spinal cord.

The fibers of each peduncle in common with the cerebral fibers have their decussation. The fibers of the *superior* cerebellar peduncles come from the dentate nuclei of the lateral bodies, decussate in the upper portion of the pons, and then go into the red nuclei which are located in the middle portion of the cerebral peduncles. From here they go into the optic thalamus and then into all parts of the cerebral hemispheres. They are efferent in function.

The fibers of the *middle* as well as those of the inferior cerebellar peduncle are afferent in function. Those of the middle cerebellar peduncle come from all parts of the cerebral hemispheres, are then transmitted by the anterior limb of the internal capsule and the foot of the cerebral peduncles into the so-called nuclei pontis in the middle part of the pons. From here begin a new set of fibers which decussate in the median raphe of the pons and then go by means of the middle cerebellar peduncles into the cortex of the lateral lobe.

The *inferior* cerebellar peduncle connects the periphery with the cerebellum by means of the spinal cord, and is concerned with unconscious muscle sense impulses. These are transmitted by the deep muscular nerves and the posterior roots into the cells of the Clark's column, where new fibers originate and ascend in the spinal cord by means of the direct cerebellar tract and then enter the cerebellum in the restiform body and go into the cortex of the vermis.

For the purpose of localization it is not necessary to know the anatomical names of the different lobules. It is, however, necessary to know that the cerebellum has a superior and an inferior surface in which are placed certain definite functions.

CEREBELLAR SYMPTOMATOLOGY.

Headache, nausea, vomiting, and choked discs are, of course, early manifestations, but only in those instances in which there is interruption in the flow of the cerebrospinal fluid, and generally indicate a lesion either in the crus or pons pressing on the aqueduct of Sylvius. Dizziness with disturbance of hearing is the result of implication of the vestibular apparatus somewhere in its course and is

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not alone a cerebellar symptom. Involvement of the motor fibers, with paresis of the limbs, increased reflexes and Babinski, indicate that there is pressure on the motor apparatus that should not be expected in lesions strictly confined to the cerebellum, except in those cases in which the lesion is very large. As a rule, motor involvement indicates a pontile lesion or a lesion in the angle pressing on the pons. The presence of cranial nerve symptoms, as of the seventh, sixth, fifth, ninth, tenth, or twelfth, indicates that the lesion is extra-cerebellar.

The chief function of the cerebellum is to synergize all movements of the body. Whatever symptoms are present, such as hypermetry,

FIG. 35.—Cases illustrating gait in cerebellar disease. Note that patient staggers in a particular direction, because the trunk inclines in that direction. Staggering to the right. (Weisenburg.)

adiadokokinesis, or tremor, result from loss of synergy. This synergy can be detected in any part, or parts, of the body. It may be present in one limb, the limbs of one side, in the upper limbs and shoulder girdle, in the lower limbs and the pelvic girdle, or in all parts of the body. Not only that, but in the development of these symptoms it is possible to tell whether the lack of synergic movement in the upper limb, for example, is in abduction or adduction, or in upward or downward movement.

In cerebellar disease, the movement, whatever it may be, is always exaggerated. For example, in a patient with a right-sided involvement, in performing a given movement, such as putting the finger to the nose or heel to knee, there is always an over-shooting of the

FIG. 36.—Staggering to the left. (Weisenburg.)

FIG. 37.—Staggering forward. (Weisenburg.)

mark. This is called *hypermetry*. In studying this movement it is important to observe not only that the finger goes beyond the nose, but that the action of the whole limb is greater than need be.

Adiadokokinesis is that symptom in which alternating movements, such as pronation and supination of the upper limbs when extended from the shoulder, are either irregular or exaggerated. In some instances the limb goes out more than it should or goes in or up or down. In others the arm will go out only, while in some it will go in, but the important point is that the movement is always exagger-

FIG. 38.—Staggering backward. (Weisenburg.)

ated, usually in a particular direction. If it should go out only and not in, it is apparent that the inward pointing center is implicated. If it should go in and not out, then the outward pointing center is involved.

The *gait* in cerebellar disease is usually described as a drunken, reeling gait. Moving picture studies show that the *gait is trunkal.* By that is meant that if a patient, for example, staggers to the right, it is the trunk which leans in that direction and the legs follow. If the patient staggers backward it is the trunk which goes back first; in other words, whichever way the body inclines the trunk goes first and the legs afterwards. There is a focal reason why a patient should stagger to the right only or to the left or forward, etc.

Trunkal movements may be of two kinds, those which implicate both *shoulder* and *pelvic girdles* and those which implicate *either the*

FIG. 39.—Staggering backward and to the right. (Weisenburg.)

FIG. 40.—Method of obtaining adiadokokinesis. Patient performs alternate to-and-fro movements. Note the left arm extends laterally more than it should. (Weisenburg.)

shoulder or the pelvic girdle alone. These can be readily recognized, for if the pelvic girdle is involved it is almost impossible for the patient to stand or walk; whereas, if the shoulder girdle is implicated, there is less difficulty in walking, the patient's trunk inclining in a given direction. If both the shoulder and pelvic girdle are implicated, there is, of course, greater difficulty in walking, but less than in pelvic girdle asynergy. It is important to recognize the various types of movement, because asynergy of the shoulder girdle indicates a lesion in the superior vermis, whereas asynergy of the pelvic girdle indicates a lesion of the inferior vermis.

Station in cerebellar disease is impaired, the patient staggering usually in the direction in which the trunk is inclined to go. There is one essential difference between the station of cerebellar disease and that of locomotor ataxia, for while in the latter the patient instinctively widens his base by separating his legs, in the former this is not necessarily so. Again, in tabes the station and gait are made worse by closing the eyes, while in cerebellar disease this does not make any difference.

In cerebellar disease *emotional expression* and *speech* are altered. Emotional expression may be lost altogether on one side in thrombosis of the superior cerebellar artery. Usually a cerebellar patient is stupid and does not have much emotional reaction. The speech is usually drawling and slow. Sometimes in cerebellar disease the head is deviated to one side, but this is not a focal symptom. Spontaneous *nystagmus* is not a cerebellar symptom inasmuch as it nearly always occurs through lesion of the bulb.

Cerebellar localization.—In brief, for the purpose of localization the cerebellum is divided into the vermis and the two lateral lobes. In the upper portion of the superior surface of the vermis have been placed the centers of the head and eyes. In the lower part of the superior portion of the vermis have been placed the centers for the upper trunk or the shoulder girdle. The movements of the lower trunk or the pelvic girdle have been localized in the inferior portion of the vermis.

The movements of the upper extremities have been placed in the superior surface of the lateral hemispheres. The movements of the right upper limb in the right lateral lobe, of the left upper limb in the left lateral lobe. These centers have been subdivided for outward, inward, upward, and downward movements. Similarly the centers for the movements of the lower extremities have been placed in the inferior surface of the lateral lobe on the right side for the right limb, left side for the left limb. They have also been subdivided for the movements into the given directions.

A *lesion therefore confined to the vermis* will cause, strictly speaking, only asynergy in the trunk; if of the superior vermis, of the move-

FIG. 41.—Method of testing hypermetry. In performing finger-to-nose test, the finger goes beyond the nose. (Weisenburg.)

FIG. 42.—Method of testing for pelvic and shoulder asynergy. Note position of patient on all fours. In this, the shoulders are still, whereas the pelvis swings to and fro, illustrating pelvic asynergy. (Weisenburg.)

ments of the shoulder girdle; if of the inferior vermis, of the pelvic girdle. Usually, however, lesions of the cerebellum are not strictly confined to the vermis, and there will be nearly always implication of the lateral lobes, in which case there will be asynergy of the move-

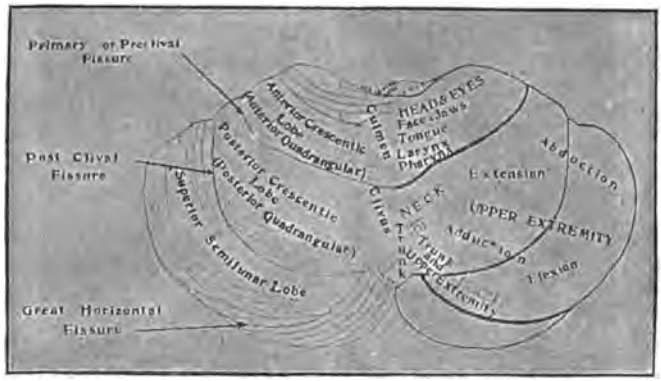

FIG. 43.—Cerebellar localization; zones and centers of the superior surface of the cerebellum. (Mills and Weisenburg.)

ment of the trunk and one or both limbs on one side, depending upon which lateral lobe is involved. In lesions of the vermis the gait and station are more commonly forward or backward and less frequently to one side.

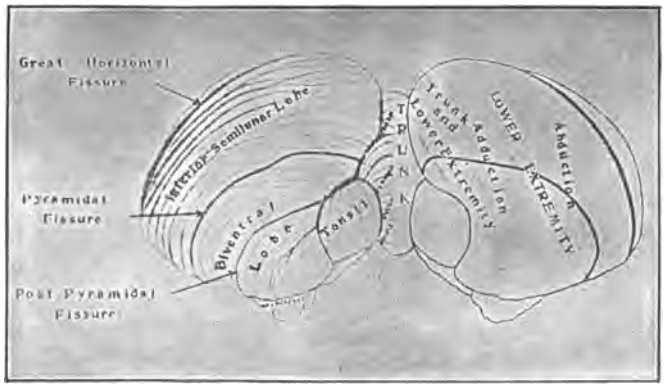

FIG. 44.—Cerebellar localization; zones and centers of the inferior surface of the cerebellum. (Mills and Weisenburg.)

Lesions of the lateral lobes cause asynergy only in the limbs on the same side. If of the right superior surface, then there will be only implication of the right upper extremity; if of the inferior surface, of the right lower extremity. A lesion of a lateral lobe will cause asynergy of the limbs of the same side, a trunkal gait in which the patient will stagger to the side of the lesion.

Lesions of the peduncles.—We possess little definite knowledge of the functions of the fibers in the cerebellar peduncles. It is supposed that the inferior and middle peduncles transmit impulses to the cerebellum, and that the superior peduncle transmits impulses from this organ. It is probable, however, that all the peduncles transmit impulses in both directions. It is also probable that the inferior peduncles are concerned chiefly with synergic movements of the lower limbs, and to a lesser extent of the upper limbs, the middle peduncle with the synergic movement of trunk and head, and that the superior peduncles are mainly concerned with the association of cerebral with cerebellar functions. All this, however, is theory, based somewhat on anatomic and physiologic facts, but not as yet supported by pathologic and clinical data.

Lesions of the cerebello-pontile angle.—The diagnosis of lesions in this area as a rule is not difficult. In this angle are found the eighth, seventh, six, and fifth cranial nerves. Tumors usually grow from the eighth nerve and in their growth gradually press upon the seventh, and sixth nerves and sometimes the fifth. If they press upon the lateral lobes of the cerebellum asynergy is obtained in the limbs on the corresponding side. Pressure upon the anterior portion of the pons implicating the motor fibers will produce motor symptoms in the arm and leg on the opposite side.

It is probable that bullet and shrapnel wounds will not involve the cerebello-pontile angle alone, but abscesses in this area secondary to middle ear disease and mastoid involvement are not unusual. It is therefore necessary to know that lesions in this area will produce first cranial nerve symptoms and secondly the result of pressure upon the cerebellum and of the pons, the symptoms depending upon the extent of the lesion.

CHAPTER II.

CRANIAL NERVES AND THEIR DISEASES.

There are twelve pairs of cranial nerves. These are known either by special names or numerically. The first and second, or the olfactory and optic, should really be considered as parts of the brain proper and not as distinct cranial nerves.

The olfactory nerve.—Numerous olfactory filaments extend from the Schneiderian membrane in the upper nasal fossæ through the cribriform plate of the ethmoid bone, where they enter the glomeruli of the olfactory bulbs. From the olfactory bulbs the lateral and olfactory tracts extend, the first directly and the latter through the stria Lancisii and fornix to the hippocampal gyrus.

In the uncinate gyrus, the dentate fascia and fornix converge. The anterior commissure may be looked upon as a commissural bundle of the olfactory bulbs. Disturbances of smell arise from lesions involving any portion of the olfactory nerve or tracts. The nerves and bulbs are often injured in fractures of the anterior fossæ of the skull, frequently resulting in loss of the sense of smell. Irritative disturbances of the smell (parosmia) are present in "shell-shock" and may sometimes form the aura of epileptic uncinate seizures.

Olfactory hallucinations occur in tumors involving the hippocampus directly or through secondary pressure. Pituitary tumors not infrequently excite uncinate fits, manifested by gustatory and olfactory hallucinations, with dreams of unreal sensations, with or without general convulsive seizures.

The optic, oculo-motor, trochlear, and abducens nerves.—These four cranial nerves will be considered together and in order, because they subserve the ocular functions.

The *optic* or *second* nerve is the nerve of vision. The course of the optic nerve fibers, their cerebral connections and lesions thereof have been discussed under the head of "Visual centers." The oculo-motor or *third* nerve has its nucleus in the back part of the cerebral peduncle on either side of the aqueduct of Sylvius. The nucleus is large, for it supplies all the muscles of the eyeball, with the exception of the external rectus and the superior oblique. In their intramedullary course the fibers of the third nerve separate and do not join until about their exit; therefore, it is possible for a basal

syphilitic lesion to cause only a partial paralysis in the distribution of the third nerve. Intramedullary lesions will, of course, produce partial ocular palsies. Lesions of the nucleus on one side are very rare, for such lesions usually are the result of an extension of tumors from the aqueduct of Sylvius, which involve both oculo-motor nuclei. The third nerve has its exit at the foot of the cerebral peduncles. Unilateral oculo-motor palsy is usually syphilitic in origin.

The *fourth* or trochlear nerve has its nucleus in the back part of the cerebral peduncle back of the third, and is very small, inasmuch as it supplies only one muscle, the superior oblique. Isolated paralysis of this muscle is rare, and is usually of transient duration and of syphilitic origin.

The *sixth* or the abducens nerve has its nucleus in the posterior portion of the pons lateral to the aqueduct of Sylvius. It has its exit at the base of the pons. The seventh nerve in its intramedullary course winds around the nucleus of the sixth and usually lesions of the pons cause combined paralysis of the sixth and seventh nerves. Paralysis of the external rectus muscle is more frequent than that of the other oculo-motor nerves, and is usually of specific origin or may result from pressure of a tumor.

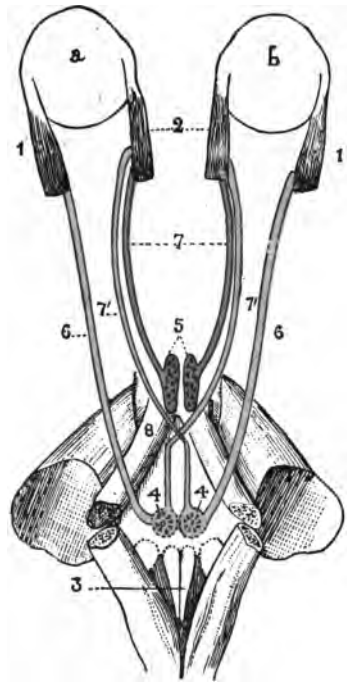

FIG. 45.—Figure showing the mode of innervation of the recti medialis and lateralis of the eye. (After Duval and Laborde.)

a, left eyeball. *b*, right eyeball. 1, rectus lateralis. 2, rectus medialis. 3, rhomboid fossa. 4, nucleus of abducent nerve. 5, nucleus of oculomotor nerve. 6, abducent nerve. 7, nerve to rectus medialis arising from the nucleus of the oculo-motor of the same side. 7′, nerve to rectus medialis arising from the nucleus of the abducent of the opposite side. 8, decussation of the fibers of the abducent nerve to the rectus medialis.

The eye and its examination.—In the routine examination of the eyes of a patient with nervous disease or injury, we wish to determine particularly the following points:

(*a*) The sensibility of the cornea.

(*b*) The size, shape, and reactions of the pupils.

(*c*) Any abnormalities of the extrinsic ocular muscles—nystagmus, spasm, squint, or paralysis.

(*d*) The acuteness of distant and near vision and the fields of vision.

(*e*) The condition of the optic disc as respects atrophy, edema, or alterations in the blood vessels.

(*f*) Other anomalies, such as exophthalmos, retraction of the eyelids, and evidences of existing or previous inflammation, injury, or operation.

The methods of conducting these examinations and the neurological significance of the results obtained will now be taken up seriatim.

Cornea.—The *sensibility* of the cornea is tested most conveniently by touching the cornea with a bit of absorbent cotton twisted into a thread. Since certain patients shrink from this test, it is well to touch the cheek first and ask the patient if he feels the cotton. Less objection will then be made to touching the cornea itself. In normal conditions when the cornea is touched the patient will wink and also give evidence of feeling some discomfort. The neurotic patient may jerk away as if he had been burned with a hot iron, but this reaction is due more to fear than to actual pain.

In other cases the winking is slow or absent, indicating hypesthesia or anesthesia of the cornea. After section of the ophthalmic division of the fifth nerve, in some cases of herpes zoster of this branch and after the instillation of cocaine, there is absolute anesthesia of one cornea, and touching it causes no winking. Again there may be merely diminished sensibility of one or both corneæ. If unilateral, this hypesthesia, without facial hypesthesia, may mean a tumor of the cerebellum on the same side. In such cases there will be also coarse nystagmus to the same side, unstable station, and muscular weakness with a tendency to fall to the same side. Or, hypesthesia of one cornea or of both may be a sign of hysteria, when corroborating symptoms may be found, such as anesthesia of the pharynx or other parts, a history of aphonia, or contraction of the fields of vision with normal pupils and optic discs.

Diminished sensibility of one cornea, therefore, means a gross lesion of the fifth nerve of that side; a lesion of the cerebellum of that side compressing the fibers of the fifth nerve, or hysteria.

The pupils.—*The pupils in health.*—The *width* of the pupils is greater in myopic individuals than in hyperopic, and greater in youth than in old age. In adolescence the average width of the pupils in moderate illumination with both eyes open is about 4 mm., one-third of the width of the cornea. In middle age the average is about 3.5 mm., while in old age it is 3 mm. or less.

The *shape* of the pupils is round in adolescence, slightly irregular in middle life, and often considerably irregular in old age.

The *reactions* of the pupils are the sensory, the direct light, the consensual light, and the convergence reaction.

The *sensory* reaction consists in a dilatation of the pupils when, for example, the skin of the temple is scratched or the skin of the neck is pinched. While it is usually well marked in early life, in middle age it is inconstant; hence its absence is of very slight diagnostic importance.

The *direct* reaction is the contraction that takes place when light is thrown into the eye observed, and the consensual is the contraction that takes place in one eye when light is thrown into the other.

In health the two pupils are always of equal size, and light thrown into one eye causes a moderate contraction of both pupils, while light thrown into both eyes causes an excessive contraction of both pupils.

The *convergence* reaction is the contraction which occurs when the eyes are brought from distant fixation into convergence and accommodation for near fixation.

The light reactions are well marked in early life, less marked in middle life, and often rather sluggish in old age, when the blood vessels are sclerosed. The convergence reaction is well marked at all ages.

The tests of pupillary reaction may be made conveniently by having the patient sit facing a window at a distance of several feet. If he is then directed to look up at the ceiling, we displace the annoying corneal reflex which ordinarily overlies the pupil and renders it a difficult matter to determine its shape. The eyes are now covered alternately to obtain the consensual light reaction, and next, while one eye is covered, the other is alternately covered and exposed to obtain the direct light reaction of each. The convergence reaction is obtained by directing the patient to fix the observer's finger held near the eyes in line with the point on the ceiling at which he has been gazing.

A finer test that will reveal slight sluggishness is that of throwing light into the eye from a distance with the ordinary ophthalmoscope. The electric ophthalmoscope is unsuited to this test. A common test is that of throwing light into the eye with a pocket electric flash light. This, however, is a coarse test that will fail to discover many sluggish pupils unless the light is held at some distance from the eye, or is so held that its rays enter the eye obliquely in order that few of the rays may fall upon the center of the retina.

The pupils in disease.—Before considering anomalies of the pupil it is well to call to mind the nerve supply of the iris. The sphincter muscle is supplied by a branch of the third nerve which, when stimulated, causes a contraction of the pupil, and when inhibited, causes a moderate dilatation. The radiating arteries of the iris, on the other hand, have their muscular coats supplied by fibers of the sympathetic nerve, which, when excited, cause a dilatation of the pupil and when inhibited, lead to its contraction.

Sympathetic pupillary disturbances.—Excitation of the cervical sympathetic causes a dilatation of one or both pupils without visual disturbances and often without the knowledge of the patient. The dilatation is usually transitory and occurs in the neurotic individual,

or following excitement, fatigue, or emotional stress. A more lasting and sometimes permanent disturbance is the contraction of the pupil, usually unilateral, that follows compression of the sympathetic fibers or center.

An injury of the spinal cord involving the cilio-spinal center which lies at the level of the lower cervical and first dorsal vertebrae, or pressure on the sympathetic fibers by an enlarged bronchial gland in tuberculosis, or pressure by cervical glands, or a goiter, may produce a unilateral complex of symptoms consisting of contraction of the pupil, narrowing of the palpebral aperture and slight sinking of the eyeball into the orbit. Here there is no disturbance of vision and the patient may not be aware of anything wrong. In doubtful cases, the instillation of 3 per cent cocain solution will confirm the diagnosis, for cocain in the normal eye excites the sympathetic nerves and causes a dilatation of the pupil and widening of the palpebral aperture. In these cases of pupillary contraction from sympathetic paralysis, cocain does not dilate the pupil or widen the palpebral aperture in the affected eye, while it has its usual effect in the other eye.

Third nerve pupillary disturbances.—Meningeal irritation may cause a spasm of the sphincter muscles with contraction of both pupils. Compression of the nucleus of the nerve for the sphincter muscle or of its fibers in the third nerve trunk will cause dilatation of the pupil. But, since the nucleus for the sphincter muscle and that for the muscle of accommodation—the ciliary muscle—lie close together and apart from the nuclei for the other muscles supplied by the third nerve, and since the fibers for the sphincter and the ciliary muscles run close together in the third nerve trunk, this paralytic dilatation of the pupil is usually accompanied by paralysis of the muscle of accommodation and the patient is unable to see near objects clearly. In these cases, too, the light reaction which is mediated by the third nerve is absent, and usually the convergence reaction as well. In the sympathetic pupillary disturbances, on the contrary, the light and convergence reactions are never abolished, although they may be somewhat reduced.

Of clinical interest are the following types:

(*a*) Paralysis of accommodation in each eye with pupils of normal size and reaction often follows diphtheria and lasts a few weeks only.

(*b*) Paralysis of accommodation and dilatation of the pupils with loss of light and convergence reactions in both eyes, without other muscular anomalies, occasionally occurs in ptomain poisoning and is likely to be permanent.

(*c*) The same condition, known as ophthalmoplegia interna, occurring in one eye only and lasting a long time, is almost invariably due to a luetic nuclear lesion.

Argyll-Robertson pupil.—Half a century ago Prof. Argyll-Robertson, of Edinburgh, described a condition in which the light reflex of the pupil is lost while the convergence reflex is preserved. He found this to be associated with anomalies of the knee-jerk, hence, with disease of the posterior columns of the spinal cord.

Our views as to the causation of the Argyll-Robertson pupil, or reflex iridoplegia, and as to its onset and course, have become very definite in recent years. Almost without exception it is a symptom of syphilis of the nervous system, and in its incipient stages it appears often within a few months after the initial infection in persons who are to exhibit frank symptoms of syphilis of the nervous system many years later. It begins with slight irregularity and inequality of the pupils, followed by sluggishness of the light reaction. In most cases the pupil gradually becomes contracted to a degree that can be explained by a spasm of the sphincter muscle only and the light reflex is entirely lost. Later, the pupils may become abnormally dilated—in some cases they are large throughout—and with this late dilatation, or preceding it, the convergence reaction may become sluggish. Vision is never affected by the onset of reflex iridoplegia alone. If near vision is affected, there is absolute iridoplegia, with an accompanying paralysis of the muscle of accommodation, namely, an internal ophthalmoplegia.

Extrinsic muscles of the eye.—Nystagmus, spasm, concomitant strabismus, and paralysis are here to be considered.

Nystagmus.—When nystagmus dates back to infancy it has no great diagnostic significance. Partial albinism or opacities of the media of the eye may be ocular causes, and difficult labor or hereditary lues may be cerebral causes, giving rise to nystagmus that is mostly constant, but increases when the eyes are moved from the primary position. A slight amount of nystagmus when the eyes are turned far to one side is fairly constant in the neurotic and the fatigued and may be called physiological.

A well marked nystagmus acquired after infancy always has significance. This may come on with the most diverse intracranial conditions, but certain types have definite significance. First, there may be nystagmus when an ocular muscle is about to be paralyzed or when it is recovering from a paralysis. This nystagmoid twitching is in the direction of the action of the muscle affected and is usually more pronounced in the affected eye than in the other. Nystagmus on looking upward often indicates a midbrain growth and precedes a paralysis of the superior rectus. Marked nystagmus in looking to one side often means a pontine lesion and precedes an associated paralysis of ocular movements to that side. Nystagmus coarse and slow to one side and fine and rapid to the other usually

means a cerebellar lesion on the side of the coarse movement. In many cases of disseminate sclerosis there is slight nystagmus in looking straight forward, which becomes exaggerated when the eyes are turned in any direction.

Spasm.—Spasmodic contraction of the orbicularis muscle or of the internal recti producing diplopia is common in hysteria. Spasm of an internal rectus with contracted pupils is often a sign of cerebral irritation, and spasm causing conjugate deviation of the eyes is usually due to cerebral irritation.

Concomitant strabismus.—When of the convergent variety, this comes on in hyperopes about the age of two years, and when of the divergent variety it comes on in adolescent myopes. There is here no limitation in the extent of mobility in either eye, but in one eye or both mobility is increased in one direction and diminished in the contrary direction. There is no diplopia. When one eye is blind or amblyopic and is not used for visual purposes, it is apt to turn inward in early life, and in adult life outward.

Paralysis.—Congenital paralysis due to hemorrhage at birth or malformation of muscles appears immediately after birth. The external rectus is the mucle most frequently involved, and a differentiation between convergent strabismus due to a birth palsy of the external rectus or to hyperopia depends often upon the immediate appearance of the former and the periodic development of the latter at about the age of two years. Next in frequency to birth paralysis of the external rectus is paralysis of one or both superior recti, sometimes accompanied by ptosis. Diplopia is not observed in these congenital cases.

Acquired paralysis of an extrinsic muscle of the eye is revealed to the patient by the existence of diplopia and to the physician by defective mobility of the eye in some direction. Tests for defective mobility, as for nystagmus, are best made by having the patient fix a bright object in the distance and then turn his head from side to side and up and down. This brings out the full excursions of the eyes to all directions and avoids the complication of convergence which comes into play when the patient fixes the finger or other near object.

If there is complete, or almost complete, paralysis of a rectus muscle, simple inspection will reveal the defect. With a slight paresis of a rectus muscle or of a superior oblique, it is necessary to cover each eye alternately, when the affected eye will be found to lag behind if moved in the direction in which the paretic muscle should move it. Thus, for example, with paresis of the right external rectus, there will be no movement of the eyes on alternate covering except when the eyes are turned far to the right, when alternate covering

will show that the right eye when covered is convergent. Similarly, with paresis of the right superior oblique muscle, the eyes will be balanced under alternate covering except when turned down and to the right, when the right eye, if covered, will be found to stand higher than the left and to be convergent. After a few weeks a paralytic condition may become complicated by contracture of the muscles antagonistic to the paralyzed muscle, when the status of the muscles must be rather laboriously worked out by holding a red glass before one eye and noting the relative position of the red and white double images of a distant light in all four quadrants of the field of fixation. This test had better be left to the trained ophthalmologist.

The position of the patient's head is often a guide to the muscle paralyzed, for if he does not close one eye to avoid diplopia he will turn his head to the right and his eyes to the left with paralysis of the right external rectus, and will turn his head down and to the right and his eyes in the contrary direction with paralysis of the right superior oblique, thus increasing the range of mobility in which he sees single.

The *site of the lesion* in ocular palsies is almost exclusively in the nucleus of the supplying nerve or in its trunk. Facial palsy and pseudo-bulbar palsies are frequently due to a supra-nuclear lesion, but since the nuclei of the ocular muscles chiefly have a cortical representation in both hemispheres, practically the only paralyses of supra-nuclear origin are an occasional ptosis and rarely a transitory conjugate deviation of the eyes.

In *order of frequency of involvement, the sixth nerve* with its long basilar course is first, then the third nerve in some or all of its branches, and finally the fourth nerve. The sixth nerve trunk is frequently involved in hemorrhages and exudations at the base of the brain, and a paralysis of both external recti alone is almost necessarily basilar. In cases of increased intracranial tension from tumor, a paralysis of one external rectus, while the other cranial nerves are spared, is quite common, but this has no localizing value since it seems to be caused by compression of the nerve trunk by a cerebellar artery and is a sign merely of general increased intracranial pressure.

In cases of *nuclear paralysis* of the muscles *supplied by the third nerve*, very frequently the sphincter of the pupil and the muscle of accomodation are spared, and the condition is then known as external ophthalmoplegia. When the intrinsic muscles are affected also, the condition is known as total ophthalmoplegia.

Nuclear lesions of the third nerve as a rule affect the recti muscles first and the levator of the upper lid later on or not at all. Compres-

sion of the nerve trunk usually causes ptosis from the beginning, and frequently the intrinsic muscles are affected as well as the extrinsic.

There are no particular signs by which a nuclear can be distinguished from a trunk *lesion of the fourth nerve*, except that here, as in the case of other cranial nerves, a trunk lesion may be preceded by meningeal headaches, whereas nuclear lesions may come on without premonitory symptoms. It is not difficult to tell whether there has been an involvement of the fourth nerve in cases of external ophthalmoplegia, because when an effort is made to look down and out, the superior oblique rotates the eyeball slightly down and out if the fourth nerve is intact, while this movement is wanting if the fourth nerve, as well as the third, is involved.

The Acuteness and the Fields of Vision.—Most affections of the optic nerve cause diminution in the acuteness or in the fields of vision, and the nature and extent of recent visual deterioration are often of as great importance in diagnosis as inspection of the optic nerve head with the ophthalmoscope.

Tests of distant vision should be made with the patient's distance glasses, if he has them, and tests for near vision with his reading glasses. If the vision for distance is below normal and he has no distance glasses, one is sometimes compelled to rely on his statements as to whether or not there has been recent failure of distant vision.

In the early forties, one's near vision fails from presbyopia, so a history of failing vision in middle age or later is of no neurological significance unless the patient is sure that it is his distant vision that has failed. A careful determination of the refraction is often desirable. In testing the illiterate or the aphasic, the Fridenberg test card, on which groups of squares graduated in size replace the usual letters, is especially useful, since the illiterate will count the squares in the group indicated and the aphasic will hold up an equal number of fingers.

Test of the Field of Vision.—When vision is very poor tests may have to be made with the pocket flashlight or with the moving hand, but with fair vision for routine purposes a white pin head of about 3 mm. is the most useful test-object. With the normal eye the field for this 3 mm. test-object has almost the same extent as that of the usual 1 cm. perimetric test-object, but defects in the field of an abnormal eye are much more readily discovered with the smaller object. When this test-object is held half way between the eye of the observer and that of the patient, 50 cm. apart, the field of each eye should be equal in extent, and if in any meridian the patient loses sight of the test-object before the observer, his field is restricted in that meridian. In testing for relative hemianopsia and for central scotoma, a small red test-object is useful, or the same 3 mm. pin head

may be used by the observer while standing several feet from the patient, who fixes the observer's eye. The greater the distance the more delicate the test.

Bjerrum devised a black cloth screen 2 m. in diameter to be viewed at a distance of 2 m. and used test-objects of white ranging from 1 to 10 mm. in diameter mounted on black rods, which are nearly invisible against the back-ground. His method is useful for plotting small or relative defects, such as central scotoma, enlargement of the blind spot, or the like. And small hand compimeters are rather more useful than perimeters in plotting defects near the point of fixation.

Considerable practice is required to plot a visual field accurately with the perimeter, because the patient becomes fatigued and unreliable in his answers, or he may not maintain proper fixation. What we wish to know for diagnosis is not so much the actual amount of contraction as the type of contraction, and this can be determined by the simpler tests.

Contraction of the field of vision, in nervous diseases, is found chiefly in four types, namely, concentric contraction, central scotoma with normal limits of the field, bitemporal hemianopsia or loss of the temporal half of the field for each eye, and homonymous hemianopsia, or loss of the right or of the left half of the field for each eye.

Concentric contraction is the rule in simple atrophy of the optic nerve such as that occurring in tabes. This same sort of field is found in beginning post-neuritic atrophy, such as that following the papilledema of brain tumor. The atrophy following papilledema is caused by the compression of the nerve fibers, and the arteries as well, by exudated material, and in its final stages we find the nasal half of the field defective just as the nasal half of the field fails in the compression atrophy of the nerve occurring in glaucoma from high intraocular tension. A concentric contraction of the field becoming smaller as the test is continued is known as the fatigue field and is a functional disturbance often associated with hypesthesia of the cornea.

Central scotoma is due chiefly to affections of the papillomacular bundle—the group of visual neurons beginning in the ganglionic cells of the macular region of the retina, passing to the temporal sector of the optic nerve head and then extending back through the optic nerve as its central or axial bundle of fibers. This bundle is particularly susceptible to compression and to poisons. Central scotoma in one eye due to pressure is found with frontal lobe tumors of the same side, and also with retained secretion in the posterior ethmoid cells in acute sinusitis. Central scotoma of toxic nature is seen in chronic tobacco poisoning, in acute wood alcohol poisoning, and in diabetes. A large central scotoma, or a number of minute scotomata

near the center, is the commonest visual field defect in disseminated sclerosis, although concentric contraction is often found with or without the central scotoma. Furthermore, hemorrhage in the macular region of the retina in cases of arterial sclerosis, causing central scotoma, is quite common.

Optic disc.—The ophthalmoscopic changes in the optic nerve head that interest us are atrophies and edemas. The *simple atrophy* or primary degeneration of the optic nerve is in the great majority of cases a symptom, early or late, of tabes. The retinal neuron first loses its medullary sheath, and later the axis cylinder and ganglion cell disappear. The spaces left are partly filled by proliferated neuroglia and the small blood vessels disappear. In consequence the entire nerve head loses its pinkish hue and grows gradually paler until finally it becomes glistening white and sharply outlined, while the retinal arteries, whose function is to nourish the ganglion-cell and nerve-fiber layers of the retina, become narrow and sclerotic.

The patient notices first a failure of acuteness of vision, and tests of the field show peripheric contraction. Central and peripheral vision fail gradually together, and after the lapse of months or years complete blindness ensues, usually in both eyes.

Pressure upon one optic tract causes a slight general pallor of each disc, as does pressure upon the chiasma by an enlarged pituitary body. In the latter case we encounter a very slight edema of the disc as well. In optic nerve affections due to ethmoid disease and to wood alcohol poisoning, there may be at first considerable edema of the nerve head, which soon passes off and is followed by the ophthalmoscopic picture of simple atrophy of the optic disc.

When the *papillomacular bundle* alone is involved, causing a central scotoma without peripheric contraction of the visual field, the inferotemporal third of the optic disc becomes pale, while the remainder of the disc retains its pinkish hue. This is the condition found in nicotine poisoning, diabetes, and in most of the optic nerve involvements of disseminated sclerosis.

Papilledema, as it occurs in the cases of brain tumor, is now generally regarded as being of a purely mechanical character. It is usually present in both eyes, but does not necessarily appear in both at the same time. The age of a process in the two eyes may be different. Optic neuritis almost similar in appearance but usually with less elevation of the disc and with more extensive involvement of the retina occurs with meningitis. This variety is of a more inflammatory nature, and the eye of the affected side of the head is involved before the other. There are special cases of chronic nephritis where the typical picture of choked disc without other changes in the retina are present, which may easily be confounded

Fig. 46.—Normal Fundus. Average Tint.

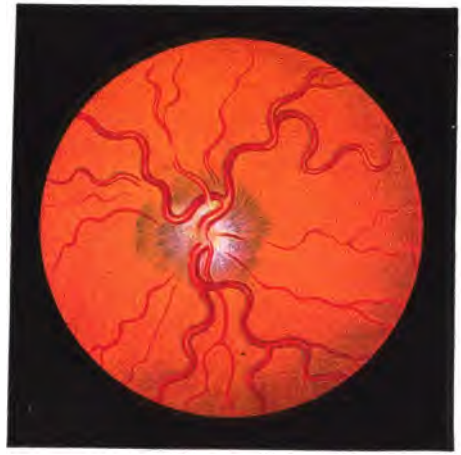

Fig. 47.—Choked Disc.

Fig. 48.—Post-Neuritic Atrophy of the Optic Nerve.

Fig. 49.—Simple Atrophy of the Optic Nerve.

Fig. 50.—Albuminuric Retinitis.

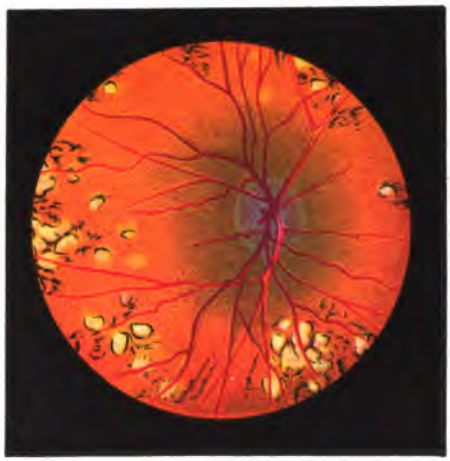

Fig. 51.—Syphilitic Choroidoretinitis.

with the findings in brain tumor. As a rule, however, the cases of choked disc in nephritis show marked involvement of the retina. Chronic intracranial pressure may be due to the tumor ends or indirectly to a blocking of the drainage channels from the ventricles, such as is seen in posterior tumors or in those blocking the iter.

Animal experimentation confirms the mechanical theory of choked disc and suggests the possibility of the tension of the eyeball by a factor in the determination of which nerve will first show edema, the group that is soft being the first to show the choking of the disc. The early or late appearance of papilledema depends much on the situation of the tumor. Tumors which interfere with the normal drainage of cerebrospinal fluid from the ventricles show pressure the earliest. Tumors at the tip of the temporo-sphenoidal lobe may by local pressure effects show early and marked changes in the disc.

The liquid in the subdural space of the brain, when forced under pressure into the subdural spaces of the optic nerve sheath, distends this space, compresses the central artery and vein of the retina which passes through it, and to some extent strangulates the nerve head. The nerve head then becomes edematous; the retinal arteries are narrowed, and the veins are dilated and tortuous from compression. After some months the nerve fibers are compressed, and atrophy of the optic nerve ensues. With the ophthalmoscope we find in the beginning a slight edema of the nerve head which veils its nasal margin. Then the upper and lower margins of the nerve head become veiled, the veins are dilated and tortuous, the nerve head and the surrounding retina become swollen with edema, and often hemorrhages appear in the retina. Some weeks after the onset, the swelling of the retina subsides to some extent and the nerve head, as a rounded, grayish, pulpy looking mass, projects one or two millimeters above its normal level. This fourth stage of papilledema may continue for months. Later a general pallor of the nerve head appears through the edema as atrophy of the nerve sets in. The swelling of the nerve head from this time subsides gradually. As papilledema does not develop in a nerve which is already atrophic, so the papilledema subsides when the nerve becomes atrophied. For months or years longer the pale nerve head has blurred margins and the retinal veins remain dilated and tortuous. In addition to the choked disc is the secondary optic atrophy resulting from the chronic intracranial pressure in cases of brain tumor. A primary optic atrophy also is seen. Primary optic atrophy in such cases of tumor results from direct pressure of the tumor upon the optic nerve and is most frequently seen in tumors of the pituitary.

Visual disturbances are noticed early. The patient complains of a cloud over his vision which annoys him. When his central vision

is tested with a well-illuminated test card it will be found to be normal or nearly so, but examination of the visual fields will show them to be defective. There may be or may not be concentric contraction of the field for white, but concentric contraction of the fields for colors is the rule. In a few cases pressure upon the chiasm, the optic tracts, or the optic radiations will add symmetrical hemianopic defects to the existing concentric contraction of the fields.

After a time central vision deteriorates. Fluctuations of vision then occur. On days when the intracranial pressure is particularly high the headache will be more severe and the sight will be more blurred. Vision in one or usually both eyes may be lost completely for a few seconds or minutes at a time, and such attacks may occur at very frequent intervals. When this stage is reached decompression can not be longer postponed. The reduction of pressure by the operation dispels the cloud before the sight and leads to enlargement of the contracted visual fields. If, however, the vision has been reduced through atrophy of the optic nerve, further atrophy may be checked by the operation, but the lost vision is not regained.

Intracranial hemorrhage may cause increased pressure with a certain degree of papilledema when the blood pressure rises as shock passes off. This papilledema is a symptom of value in confirming the existence of increased intracranial pressure, but in itself does not cause disturbances of vision or require cerebral decompression for its relief.

In the present war many cases of gunshot injuries of the vault, resulting in fissured, grooved, or depressed fractures, or in those associated with fracture of the inner table alone, have been associated with papilledema. This has been observed in about 60 per cent of the cases, but the swelling is usually not over 2 D., although in some cases the intense choking such as occurs in the brain tumor has been observed. Vision may or may not be affected. Symptoms of cerebral compression are generally present, and the disc manifestations, as a rule, promptly subside after the relief of the intracranial tension.

The trigeminal.—The trigeminal nerve is a mixed sensory and motor nerve. The sensory fibers take their origin from the Gasserian ganglion. The sensory root of the ganglion enters the brain stem at the middle of the pons. The peripheral branches are three in number:

(1) The ophthalmic division, which enters the orbit by the sphenoidal fissure.

(2) The superior maxillary division, which passes through the foramen rotundum, across the sphenomaxillary fossa, and through the infra-orbital canal and appears at the infra-orbital foramen.

(3) The inferior maxillary division, which leaves the skull through the foramen ovale.

The motor root runs with the inferior maxillary division. The trigeminal nerve supplies sensation to the skin of the face, conjunctiva, cornea, mucous membranes of nose, mouth, tongue, soft palate, and the teeth.

The area of skin supplied by the trigeminal and its various branches may be seen in diagram (Fig. 52). It is important to note that a considerable portion of the skin over the lower jaw is not supplied by the trigeminal but by the upper cervical nerves.

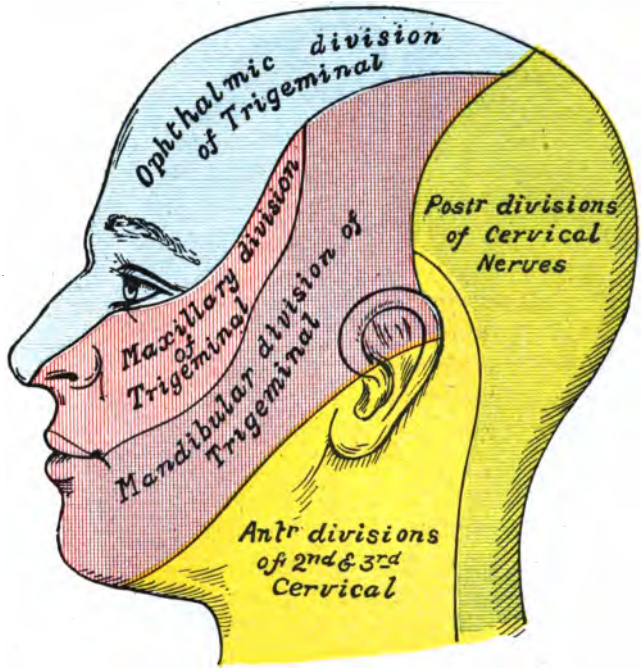
Ophthalmic division of Trigeminal
Maxillary division of Trigeminal
Mandibular division of Trigeminal
Postr divisions of Cervical Nerves
Antr divisions of 2nd & 3rd Cervical

FIG. 52.—Diagram showing cutaneous areas of face and scalp.

The motor supply includes the muscles of mastication: masseters, temporals, and pterygoids. To test for paralysis of masseters and temporals, the examiner places his hand over the corresponding muscles and notes the presence or absence of contraction on biting. The pterygoids are tested by noting any deflection of the jaw on opening the mouth. The jaw is deflected toward the side of the paralyzed muscle.

Complete division of the fifth could occur only from a lesion at the base of the brain. Wounds of the face may involve one or more branches. If the inferior maxillary division is injured close to its exit from the skull, the muscles of mastication will be paralyzed. An irritative syndrome has been described somewhat similar to causalgia, following wounds of the eye with partial amputation of the eyeball.

The facial nerve.—The *facial or seventh nerve* supplies the muscles of the face. Its nucleus is in the lower and lateral portions of the pons, and the nerve in its course outward surrounds the nucleus of the sixth nerve. Its exit is just between the pons and medulla. Because of this anatomic relation, any gross lesion involving the seventh nucleus will nearly always implicate the sixth, and vice versa. The usual form of facial palsy is that known as peripheral or Bell's palsy. Lesions causing this may be either in the pons, at the exit of the

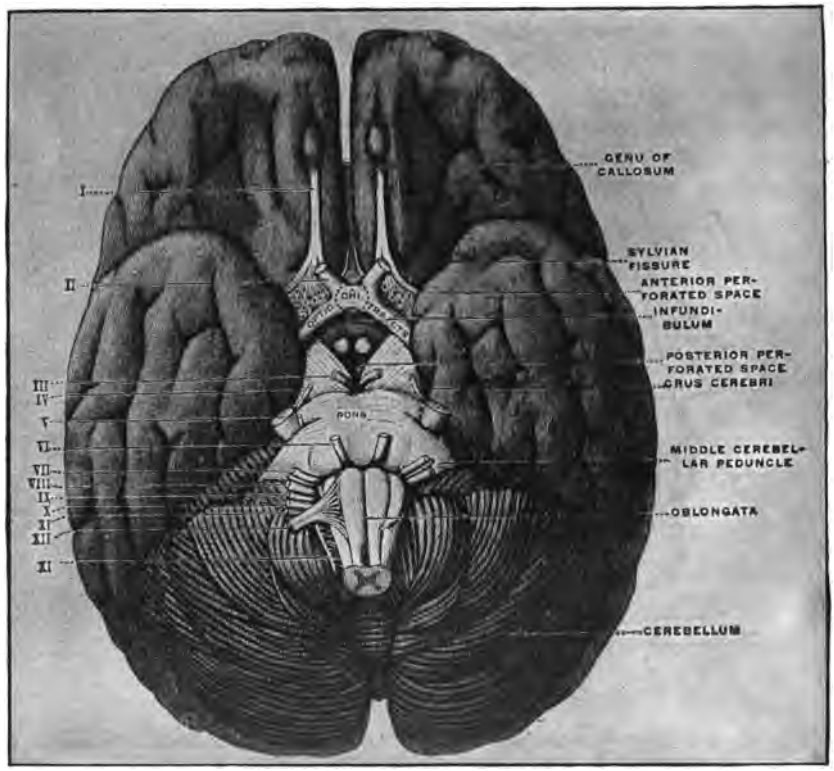

FIG. 53.—Undersurface of the brain, showing the superficial origins of the cranial nerves. The Roman numerals indicate the nerves. (Testut.)

nerve at the base of the brain. in the Fallopian canal, or in its extracranial course.

Central facial palsy is that form of facial paralysis in which the lower part of the face only is paralyzed, and is the result of a lesion in any portion of the central facial fibers between the facial centers in the cortex and its nucleus in the pons, as, for instance, in a capsular hemiplegia. The reason for the escape of the upper portion of the face in such paralysis is because wrinkling of the brow, in common with other bilateral functions, such as chewing, eating, and swallowing, has bilateral cortical innervation, and to cause paralysis of such functions there must be bilateral cerebral lesions.

In a large majority of cases ordinary peripheral facial palsy is the result of a neuritis which may be of rheumatic origin or may be part of a poliomyelitis. Rarely it is due to basal syphilis, tumors, fractures, etc. When resulting from a lesion in the pons, facial paralysis is generally accompanied by other symptoms, such as palsy of the sixth nerve, paralysis of associated ocular movement, or hemiplegia upon the other side. Abscess of the middle ear is a common cause, as well as mastoid operations. When the lesion is in the Fallopian canal, we have, in addition to the usual symptoms, temporary disturbance of taste in the anterior two-thirds of the tongue because of involvement of the chorda tympani which runs along with the seventh nerve in the Fallopian canal.

The symptoms of peripheral paralysis of the facial nerve depend upon the degree of its involvement. When the paralysis is total, there is inability to wrinkle the brow, to shut the eye, to elevate the corner of the mouth, to whistle, or to pronounce labials properly. Besides, there will be drooping of the lower lid and of the corner of the mouth, and the wrinkles on that side of the face will be smoothed out. Because of the drooping of the lower lid there will be widening of the palpebral fissure, and excessive flow of tears because of the lack of proper conduction into the nasal cavity. Electrical reactions of degeneration will be found, their degree depending upon the extent of the neuritis. Sensory disturbances may be present at the onset, when the patient may complain of pain in the face, and there may also rarely be herpetic eruptions in the ear because of involvement of the geniculate ganglion.

Most cases of peripheral facial paralysis recover, provided the cause is an ordinary neuritis and prompt treatment is instituted. In those cases in which the nerve is cut, unless an anastomosis is performed recovery can not be expected.

The eighth nerve.—The eighth cranial nerve is not an anatomical entity but is made up of two distinct parts, the cochlear nerve, which has to do solely with the function of sound perception, and the vestibular nerve, which has to do with orientation in space and the preservation of the equilibrium of the body. A destruction of the peripheral end organs of the eighth nerve in the labyrinth of the ear not only produces a disturbance in the function of hearing but gives rise, as well, to disturbances in equilibrium, vertigo, nystagmus, and often nausea and vomiting. The symptom of vertigo is now recognized to be as distinctly a labyrinth symptom as is tinnitus aurium, and when present indicates a disturbance of the vestibular mechanism either in the peripheral apparatus in the internal ear or in the central pathways of the vestibular nerve. An appreciation of this fact has increased very greatly the interest of the neurologist in otology, especially as it relates to disturbances arising from disease of the internal ear and

to the diagnosis of intra-cranial disorders. The following pages, therefore, are devoted to a brief presentation of those facts in the anatomy, physiology and pathology of the internal ear which are essential for a clear understanding of the phenomena associated with disturbance of the eight nerve, either in the peripheral apparatus in the internal ear or in its central pathways.

Anatomical.—The organ of hearing is made up of two distinct mechanisms. The one, which is encased in the labyrinth or internal ear, has to do with the perception of sound impulses. The other has to do with the conduction of sound impulses into the labyrinth, and is known as the sound-conducting mechanism. It is made up of the

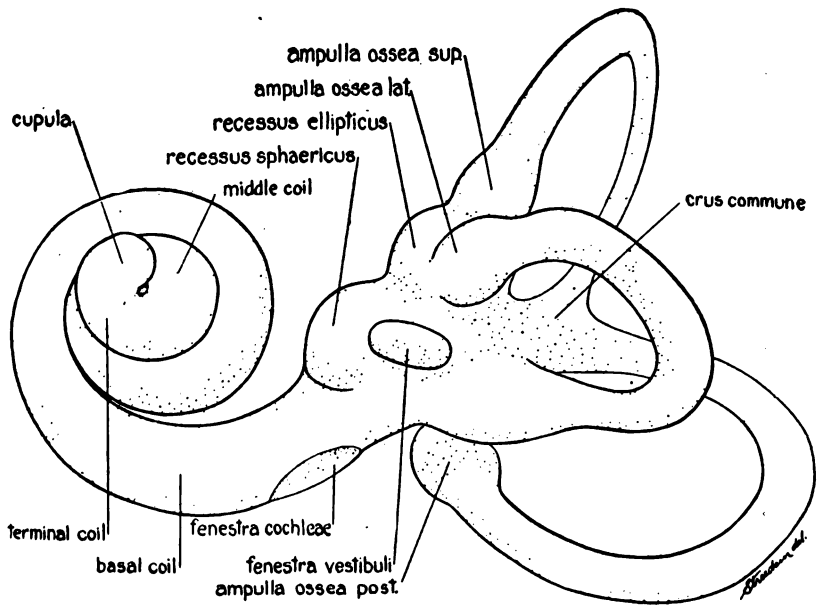

FIG. 54.—Sketch of a Wood's metal cast of the bony cavities of the labyrinth, showing the oval window for the articulation of the stapes, the recessus sphericus for the lodgment of the saccule, and the recessus ellipticus for the utricle, the cochlea with the round window and the three semicircular canals with their ampullae. (Shambaugh.)

external ear and the middle ear. The internal ear is encased in the petrous portion of the temporal bone. A complicated series of cavities which make up the osseous labyrinth consist of a central chamber, which we call the vestibule, a tubular prolongation from the anterior wall of the vestibule, which in mammals is coiled upon itself in the manner of a snail shell and which is designated the cochlea, and finally, a series of three arched canals from the end of the vestibule opposite the cochlea, which are called semicircular canals. A cast of these cavities, as shown in Figs. 54 and 55, gives one a clear idea of their makeup.

The *cavity of the vestibule* communicates with the middle ear through the "oval window." In this window the foot-plate of the stapes is attached. The vestibule communicates also with the posterior brain fossa by means of an opening on the posterior aspect of the petrous bone. This passage is called the aqueductus vestibuli.

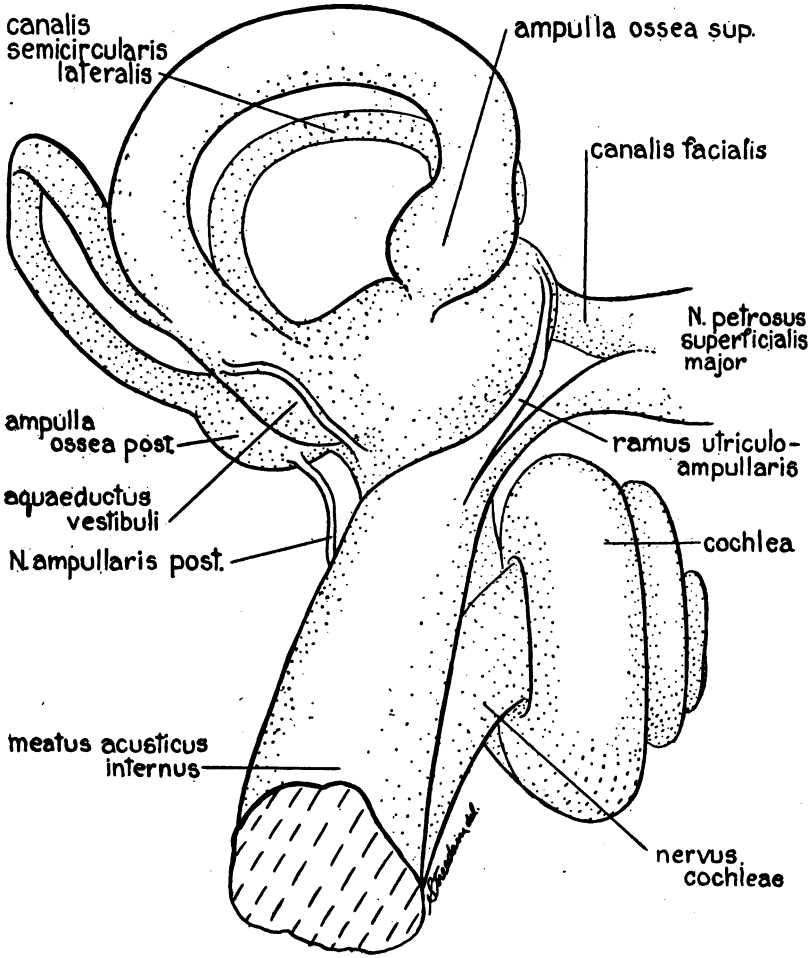

FIG. 55.—Wood's metal cast of the labyrinth, with the channels for the branches of the eighth nerve to the several end-organs in the labyrinth. Note the cochlear nerve entering the modiolus at the base of the cochlea, the ramus utriculo ampullaris of the vestibular nerve which supplies the macula acustica of the utricle and the crista acustica of the horizontal and superior semicircular canals, also the passage of a branch of the vestibular nerve to the crista of the posterior semicircular canal. The canal for the facial nerve is also shown where this nerve joins the great superficial petrosal to form the trunk of the facial nerve as it passes out through the temporal bone. (Shambaugh.)

There are two depressions or recesses on the walls of the vestibule. One, the recessus ellipticus, contains a part of the membranous labyrinth known as the utricle. The other, the recessus sphericus, houses another part of the membranous labvrinth known as the saccule.

The tube forming the *cochlea* projects from the anterior wall of the vestibule. The apex of the cochlea lies close to the tympanic orifice of the Eustachian tube. The cochlea communicates with the middle ear through the "round window." This window is closed by a strong membrane, the membrana tympani secundaria. The first turn of the cochlea forms a conspicuous prominence on the inner wall of the tympanum, between the oval and round windows. This prominence is known as the promontory. The cochlea communicates with the posterior brain fossa by means of a small tube, which begins at a point on the under surface of the basal coil close to the vestibule, and ends in a depression on the posterior aspect of the petrous bone, midway between the internal meatus and the bulb of the jugular. This tube is known as the aqueductus cochleæ and furnishes a means of communication between the fluid filling the osseous labyrinth, the perilymph, and the fluid found in the subarachnoid space. The main channel for the exit of veins from the labyrinth is along this aqueductus cochleæ. The coils forming the cochlea encircle an osseous cone called the modiolus. This is perforated by numerous channels for the filaments of the cochlear nerve and its spiral ganglion, as well as for blood vessels and lymphatics of the cochlea. A cross section through the cochlea discloses a bony shell projecting from the modiolus part way across the tube throughout its entire length. This is called the lamina spiralis ossea. At the base of the cochlea it forms a relatively broad shelf, which gradually becomes narrower toward the apex.

The *three semicircular canals* known respectively as the *horizontal*, the *superior* and the *posterior canals*, are placed so as to occupy the three planes in space. The horizontal canals on the two sides occupy, in man, approximately, the horizontal plane. The superior canal on one side occupies the same plane as the posterior canal on the opposite side. Two of the canals come into close relation with the cavities forming the middle ear. The horizontal canal forms a prominence on the floor of the passage connecting the tympanic cavity with the antrum. The superior canal lies just above the horizontal in the internal wall of the same passageway. The posterior canal alone is placed remote from the middle ear. Each canal has at one end, just where it joins the vestibule, a bulbous enlargement called the ampulla. In the case of the horizontal and superior canals this ampulla is on the anterior crus, and the posterior canal has its ampulla on the posterior crus. The walls of the osseous labyrinth are lined with an endosteum and the cavities are filled with fluid called perilymph, which communicates through the aqueductus cochleæ with the fluid in the subarachnoid space.

Suspended in the *perilymph* is the membraneous labyrinth, which is an epithelial structure derived from the ectoderm. In the vesti-

bule this membraneous structure consists of two bags, known as the saccule and the utricle. The two are connected by a tube from each, which unite to form a duct, known as the ductus endolymphaticus,

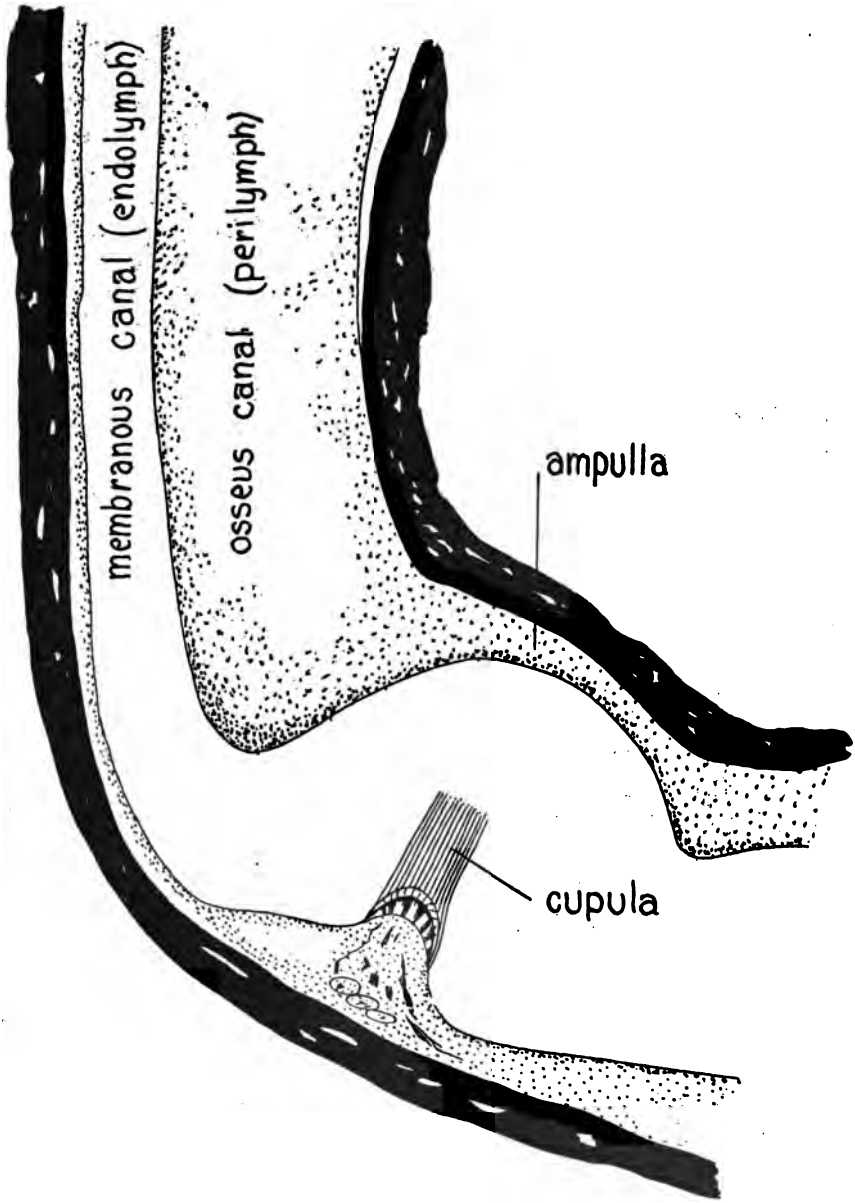

FIG. 56.—Part of a semicircular canal, showing the relation of the osseous and membranous canals and the osseous and membranous ampulla with its crista acustica. (Shambaugh.)

which passes out of the osseous labyrinth by way of the aqueductus vestibuli. Where the aqueduct emerges on the posterior surface of the petrous bone, the ductus endolymphaticus spreads out between

the layers of the dura into a closed bag, known as the saccus endolymphaticus. The membraneous labyrinth in the cochlea forms a tubular prolongation called the ductus cochlearis. This tube begins at the point where the basal coil joins the vestibule and ends at the apex of the cochlea. The ductus cochlearis communicates with the saccule through a small tubule called the ductus reuniens. In the semicircular canals the membraneous labyrinth follows more or less closely the outlines of the osseous canal (Fig. 56). It consists of a tube called the ductus semicircularis, attached along the convex wall of the bony canal, and a bulbous enlargement, a membraneous ampulla, which fits into the osseous ampulla. The membranous canals open freely at each end into the utricle. The membranous labyrinth is composed, therefore, of a series of cavities which communicate

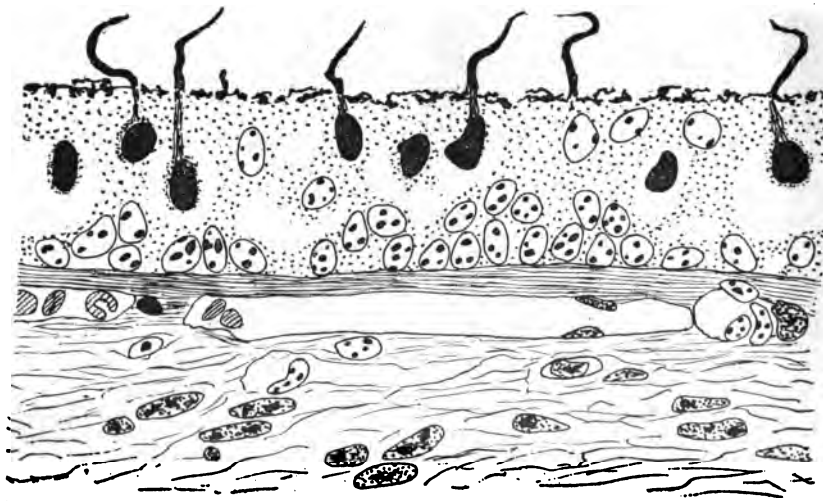

FIG. 57.—Section through the end-organ in the vestibule, the macula acustica. Note the peculiar hair-bearing cells. The otolith membrane is not shown in the drawings. It lies above the hair cells, the hairs of which are attached to its undersurface. (Shambaugh.)

with each other and form as a whole a closed space which is filled with a fluid, the endolymph.

Three types of special sense organs are located within the inner ear. The first type is found in the vestibule and is known as the macula. (Fig. 57.) Of these, there are two, one for the saccule, one for the utricle. The second type is found in the cochlea and is called the Organ of Corti. (Fig. 58.) It is attached to a membrane which stretches between the free margin of the lamina spiralis ossea and the outer wall of the cochlear tube. This is known as the membrana basilaris. The third type of end-organ is located in the ampullæ of the semicircular canals and is known as the crista. (Fig. 59.) Of this type there are three in all, one for each canal. These three types of end-organs appear superficially quite different, but when

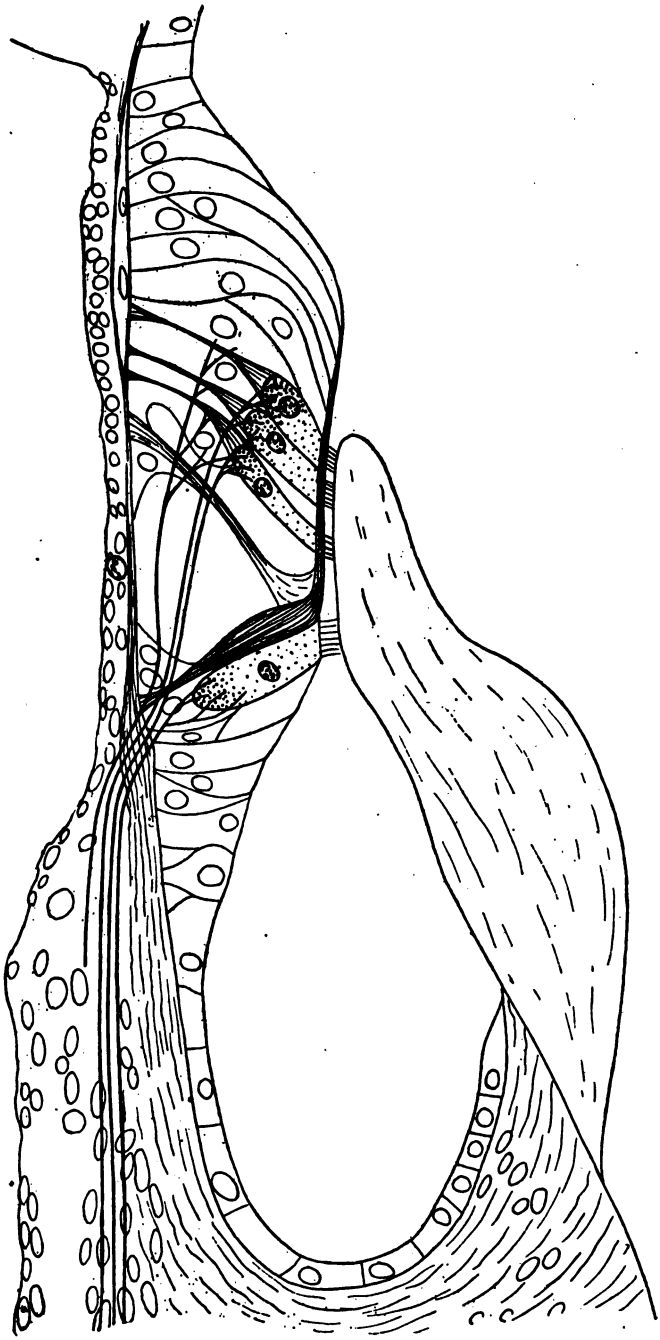

FIG. 58.—The Organ of Corti, from the cochlea of a newborn pig. Note the relation of the hairs of the hair cells to the undersurface of the membrana tectoria, to which they are normally attached, the membrana basilaris, with its blood vessels directly under the tunnel of Corti. The terminal filaments of the cochlear nerve have been added in a semi-schematic fashion. (Shambaugh.)

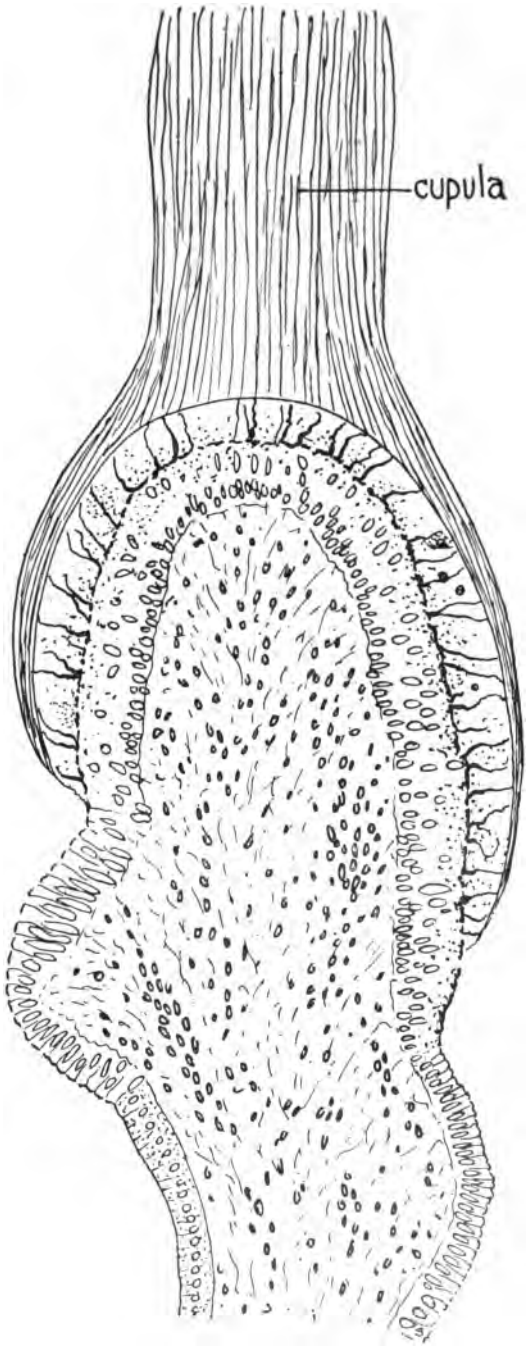

FIG. 59.—Cross section of the crista acustica of the horizontal semicircular canal of a newborn pig. Note the relation of the hairs of the hair cells to the undersurface of the cupula. On the side of the crista toward the canal the area of hair cells is more extensive than on the side toward the utricle. The greater response is elicited by the impact of an endolymph current against the side of the crista toward the canal. (Shambaugh.)

examined closely are found to be fundamentally alike in their structure. In each end-organ the important physiological element is a peculiar hair-bearing cell which receives the terminal filaments of the eighth nerve, and in which the transference of a physical to a nervous impulse takes place. In each end-organ, moreover, there is a peculiar structure, also of epithelial origin, which overhangs the hair cells and into the undersurface of which, in each instance, the hairs of the hair cells are attached. In the macula acustica this structure is known as the otolith membrane, in the Organ of Corti it is the membrana tectoria, and in the crista acustica it is the cupula. This fundamental similarity in the structure of the three types of end-organs found in the labyrinth gives an important clue, as will be pointed out under "Physiology of the Labyrinth," to the way in which these end-organs respond normally to stimuli.

Physiological.—The *labyrinth* of the ear is not solely an organ of hearing, as was formerly supposed, but in addition to its function as the sound-perceiving mechanism, it fills an important role as the end-organ for preserving the equilibrium of the body. The part of the labyrinth concerned in the function of hearing is the cochlea, whereas the vestibule and the semi-circular canals constitute a mechanism which gives us knowledge of our position in space, our sense of turning motion, and assists in preserving the equilibrium of the body. The eighth nerve, instead of being an anatomical entity, as is the optic nerve, for example, consists of two separate nerves, the cochlear and the vestibular, each having its own distinct central origin and distinct peripheral termination. The cochlear nerve alone has to do with the function of hearing and is distributed to the haircells of Corti's organ. The vestibular nerve has nothing to do with the function of hearing, and is distributed to the end-organs of the vestibule and semi-circular canals, the maculæ and the cristæ.

The *physiology of hearing* has formed an interesting subject for speculation since the writings of Aristotle, in the third century B. C. Sound perception has been attributed to the cochlea as long as there has been any knowledge regarding the existence of this structure. The fundamental conceptions in the various speculations upon the physiology of the cochlea, at least since the middle of the seventeenth century are these: First, that the perception of tones is dependent on the sympathetic vibration of structures found in the cochlea; second, that the perception of tones in different parts of the scale takes place in separate and distinct parts of the cochlea. Before the discovery of the existence of the membranous labyrinth and of the organ of Corti, the vibrating mechanism in the cochlea which responded to the impulses of sound waves was supposed to be the lamina spiralis ossea, and since this bony shelf was found to be much broader in the basal coil than at the apex of the cochlea, it

was assumed that the perception of low tones took place in the basal coil and the higher tones near the apex of the cochlea.

Since the discovery of the organ of Corti, in 1851, it has been generally accepted that tone perception results from the stimulation of the hair cells in this organ. Helmholtz accepted the prevailing conception that perception of tone impulses was dependent on the sympathetic vibration of structures in the cochlea, and attempted to apply this to the organ of Corti. The actual stimulation of the hair cells is evidently due to an interaction between the projecting hairs and the membrana tectoria. The real relation between these hairs and the membrana tectoria was not understood when Helmholtz did his work, for it was believed that the two are normally separated by an appreciable space. In order, therefore, to bring about the contact between the membrana tectoria and the projecting hairs, Helmholtz assumed that the basilar membrane was the vibrating mechanism which responded in the several parts of the cochlea to tones of different pitch, and in this way brushed groups of hair cells in different parts of the cochlea, depending on the pitch, against the membrana tectoria, and thus accomplished the stimulation of different groups of hair cells for each distinct tone.

More recent anatomical investigation has demonstrated that the hairs of the hair cells of Corti's organ are normally in actual contact with and adherent to the under surface of the membrana tectoria, exactly as are the hair cells of the macula and the crista in contact with the otolith membrane and cupula respectively. It has been shown, moreover, that the basilar membrane does not possess physical properties which would make it possible for it to act as a vibrating mechanism. For example, the blood vessel (see Fig. 58) attached to the under surface of the basilar membrane makes it impossible for it to vibrate the same at all times to the same impulse, a fundamental requirement of a vibrating mechanism. The close analogy, as pointed out above, in the construction of the three types of end-organs found in the labyrinth, makes it clear that the stimulation of the hair cells is in each instance accomplished in the same way, namely, by the movement of the superimposed structure against the hairs; that is, by the movement of the otolith membrane, the cupula, and the membrana tectoria. In order to account for the various phenomena associated with tone perception it is necessary to assume that different tones in the scale produce vibrations in different parts of the membrana tectoria, accomplishing in this way the stimulation of a different group of hair cells for each particular tone. The perception for the tones at the upper end of the scale takes place apparently in the basal coil, where the membrana tectoria is quite small, and the low tones near the apex of the cochlea, where this structure is relatively larger.

The *fundamental facts in the physiology of the semicircular canals* are quite simple, and the phenomena resulting from the stimulation of the crista are now readily understood. The following is a brief statement of these facts. The normal stimulation of the crista is the result of the impact of an endolymph current against the sides of the cupula. Only those hair cells located on the side of the crista receiving the impact of the endolymph are stimulated. It follows, therefore, that a different group of hair cells of the crista is stimulated by a current in one direction in a semicircular canal than by an endolymph current in the opposite direction. Endolymph currents in the semicircular canals give rise to forced movements, which are always in the plane of the canal being stimulated. An endolymph current in one direction in a canal produces movements toward one side, while a current in the opposite direction in the same canal produces movements toward the opposite side. The forced movements resulting from an endolymph current are always in the direction of this current; that is, from right to left or from left to right, as the case may be. An endolymph current in one direction in a canal always gives rise to a more vigorous response than that elicited by a current in the opposite direction. This means that the hair cells on one side of a crista are more sensitive to stimuli than are those on the opposite side of the same crista. The more sensitive group in each canal is the one which, when stimulated, produces movements toward the opposite side. In the horizontal canals it is the hair cells on the side of the crista toward the canal that produce the greater response, whereas in the superior and posterior canals it is the cells on the side of the crista toward the utricle that are the more sensitive. Fig. 60.

When we associate the fundamental reactions following stimulation of the hair cells of the cristæ with the fact that the skeletal muscles are normally under the influence of constant tonus impulses emanating from the hair cells of the cristæ, we have at once a plausible explanation for the disturbance in equilibrium resulting from a unilateral disturbance in the function of the semi-circular canals. It is clear from the facts already given that the stronger tonus impulses from each canal will be those which produce movements toward the opposite side. The effect of the tonus impulses from the right labyrinth will be, therefore, to produce forced movements toward the left, and from the left labyrinth toward the right. So long as the tonus impulses from the two labyrinths are equal, a state of equilibrium is preserved. A depression in the function of one labyrinth will result in the tonus impulses from the opposite labyrinth, producing forced movements toward the affected side. The patient will tend, therefore, to fall toward the affected side. The eyes, too, will be drawn forcibly toward the affected side, but will be quickly drawn

back again toward the normal side. The nystagmus set up in this way will have the quick component directed toward the normal side. The disturbance in equilibrium resulting from a depression of function in one labyrinth is always temporary, since a readjustment of the tonus impulses so as to bring about a state of equilibrium soon takes place. When the unilateral loss of function proceeds slowly enough, it may proceed to the point of complete suppression without at any time giving rise to vertigo or nystagmus. A unilateral disturbance in tonus, in order to produce vertigo or nystagmus must necessarily be a sudden disturbance.

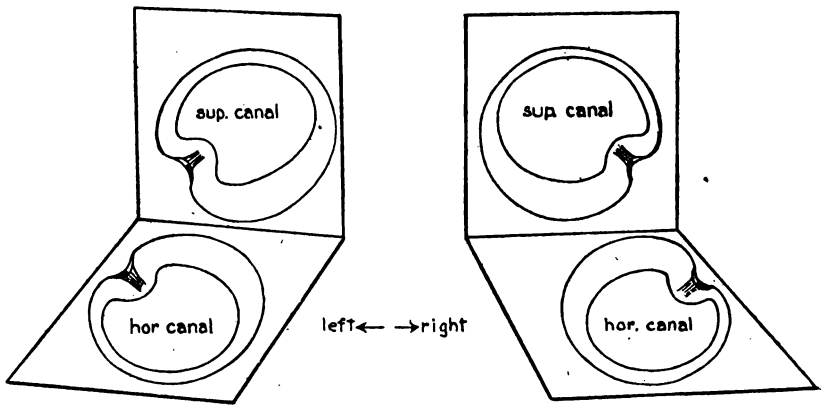

FIG. 60.—Schematic drawing showing the horizontal and superior canals on the two sides. The more sensitive side of each crista is represented dark. In the horizontal canal the more sensitive hair cells are on the side of the crista toward the canal, whereas in the superior canals the more sensitive cells are on the side of the crista toward the utricle. The posterior canals are not represented. They react similarly to the superior canals. Since it is known that the hair cells stimulated by an endolymph current affect the muscles which cause movements in the plane of the canals and in the direction of the endolymph current, from a glance at this drawing it is apparent that the stronger impulses emanating from the hair cells of each crista will be those which produce movements toward the opposite side. A destruction of one labyrinth with the unilateral suppression of labyrinth tonus will produce a nystagmus with the quick component directed to the opposite side while the patient will be inclined to fall toward the affected side, as the result of the action of tonus impulses emanating from the normal labyrinth. (Shambaugh.)

Pathological.—*Disease of the internal ear* has come to be a subject of special interest, because of the symptoms of vertigo and disturbed equilibrium which result from pathological changes involving the vestibular apparatus. Previous to the appearance of the writings of Ménière, in 1861, vertigo had always been looked upon as a symptom pathognomonic of intracranial disease. As early as 1828, Flourens, to be sure, had called attention to the peculiar disturbance in equilibrium resulting from injuries to the semicircular canals in pigeons. These phenomena, however, were explained by some as the result of subjective *sound* impressions due to injury to the sound-perceiving

apparatus, as the entire labyrinth at that time was supposed to be. Others explained these disturbances as the result of injury to the brain incidental to the operations on the semicircular canals. Ménière attributed the symptoms observed in his cases—the sudden onset of deafness, with tinnitus and vertigo—to the rupture of blood-vessels in the labyrinth. Since the publications of Ménière, medical literature is filled with reports of cases presenting the *Ménière symptom-complex*, which have been classified as cases of "Ménière's disease." As a matter of fact, while primary disease of the internal ear is a frequent cause for this symptom-complex, it is now quite evident that actual hemorrhage into the labyrinth can be responsible for these symptoms in only a small percentage of cases. On the other hand, it is quite apparent from the facts discussed under "Physiology of the Labyrinth" that any sudden disturbance of function in the vestibular mechanism will give rise to vertigo. This may be the result of disease of the labyrinth occurring primarily, or as the result of the extension of infection from the middle ear or the brain cavity; or, finally, the symptoms may be the result of disease affecting the intracranial pathways of the vestibular nerve.

Traumatic injury of the labyrinth.—The most typical cases of *vertigo* resulting from sudden disturbance of the function of the internal ear are cases of traumatic injury of the labyrinth. Such injury results not infrequently from a fracture of the base of the skull which passes through the petrous portion of the temporal bone and produces a sudden destruction of the function of the internal ear. *Injuries to the labyrinth* may occur in the course of a mastoid operation, either by chiseling through the horizontal canal where it makes a prominence in the floor of the aditus ad antrum, or by dislocation of the stapes during a too vigorous currettement of the tympanum. In any case, the *symptoms* are the same, a sudden and complete destruction of the function of the labyrinth. The deafness is associated for a time at least with a more or less annoying tinnitus. The unilateral suppression of labyrinth tonus gives rise to a marked disturbance in equilibrium, the patient is not able to stand, but falls to the affected side as the result of forced movements produced by the tonus impulses emanating from the normal labyrinth, acting now without the restraint from opposing impulses from the destroyed labyrinth. Not only is the patient not able to stand, but he experiences great annoyance from sensations of vertigo, associated with nausea and vomiting. A violent nystagmus is produced, the eyes are drawn forcibly toward the affected side, followed by a quick recoil toward the normal side as the result of cerebral cortical impulses. When the patient is unconscious these cortical impulses are absent, and the eyes remain fixed in their deviation

toward the affected side. The eye movements are the result of impulses emanating from all three canals, and the resulting movements are therefore rotary in character. The intensity of the nystagmus and the annoyance from the sensation of vertigo are increased by looking toward the side of the quick component, and are diminished by turning the eyes toward the side of the slow component, that is, toward the affected side. For this reason, such a patient will always be found lying by choice on the normal side, so that when he looks out, the position of his eyes will tend to diminish his subjective annoyance. The intensity of the symptoms of disturbed equilibrium, vertigo and nystagmus soon begin to diminish, so that after a very few days a marked improvement can be noted. It is usually only a couple of weeks in such cases until the nystagmus and active vertigo disappear. A more or less unsteadiness in walking is all that may be apparent from the disturbance in equilibrium, and this, too, disappears after the lapse of a few weeks or months.

Labyrinth disease secondary to middle ear infection.—*Inflammatory disease of the middle ear* may invade the labyrinth and give rise to either a circumscribed or a diffuse labyrinthitis. The circumscribed labyrinthitis occurs only as a complication of chronic suppurative otitis media. It is the result of a gradual erosion through the bony capsule of the labyrinth, and the production of a labyrinth fistula. The part of the labyrinth contiguous to the fistula becomes walled off by granulations, and there is produced what we call a circumscribed labyrinthitis. Such cases may experience, from time to time, perhaps as the result of an acute exacerbation of the otitis media, some vertigo associated with nystagmus. As a rule, no spontaneous symptoms of disturbance of the vestibular apparatus are evident. The fistula test (compression of air in the external canal by means of a Politzer bag) produces active nystagmus and vertigo. Since the fistula is usually located in the horizontal canal, where it lies exposed in the floor of the aditus, compression of air will produce a flow of endolymph from the canal toward the ampulla, and the forced movements, therefore, will be in the horizontal plane, and directed toward the normal side. The quick component of the nystagmus will thus be directed toward the affected side. On reversing the process, that is, by producing suction in the external canal, the flow of endolymph will be in the opposite direction, that is, from the utricle toward the ampulla, and now the direction of the slow component of the nystagmus will be toward the affected side, with the quick component toward the normal side.

Diffuse labyrinthitis secondary to middle ear infection occurs as a complication of acute as well as of chronic otitis media. When

it complicates a chronic discharge from the middle ear the labyrinthitis usually develops as the result of an acute exacerbation of the chronic process. The inflammatory changes in the labyrinth occurring in cases of diffuse labyrinthitis show as wide a variation in their severity as do the inflammatory changes which occur in the middle ear in cases of acute otitis media. In the milder cases the inflammatory changes in the labyrinth are limited to a congestion associated with the outpouring of a serous exudate. These changes are quite analagous to the reactions in the tympanum in mild cases of acute otitis media. Between such cases of mild serous labyrinthitis and those of diffuse purulent labyrinth infection there occur all shades of varying severity. Just as in cases of acute otitis media we can not always make a sharp distinction between those where the exudate in the tympanum is purely serous and others where it is more muco-purulent, or even entirely purulent, so in cases of labyrinthitis we can not always make clinically a sharp differentiation between the cases where the exudate in the labyrinth is serous, sero-purulent, or entirely purulent. The disturbance in the function of the labyrinth which occurs in these cases varies between partial temporary depression of function and a complete but temporary loss in function to the cases of complete and permanent destruction of function. As a rule, the cases where this depression of function is but temporary are the cases where there has never been more than a partial loss of function. On the other hand, in most of the cases where the reaction in the labyrinth has been severe enough to produce a complete suppression of function this loss of function remains permanent. It is evident that the milder cases of labyrinthitis, those where the inflammatory exudate in the labyrinth is of a serous character, are the cases where there is but a partial depression of function, and that the cases of diffuse suppuration in the labyrinth probably always result not alone in a complete suppression of function but in a permanent loss of function as well. On the other hand, we are not able to assume that all the cases where there is complete suppression of function are cases of purulent labyrinthitis. In some cases where the suppression of function seems to be complete, there is later, at least, a partial return of function. Such cases we believe are not cases of purulent, but, rather, the more severe cases of serous labyrinthitis.

The *clinical differentiation between cases of purulent labyrinthitis and cases of serous labyrinthitis* is of importance, not only because of the prognosis regarding function of the labyrinth but more because the severe cases of labyrinthitis lead not infrequently to fatal intracranial complications, in the form of meningitis or cerebellar abscess. In these cases the prompt opening of the labyrinth is often

indicated in order to prevent the possibility of such an intracranial complication.

From this discussion certain conclusions are apparent. One is that cases of *serous labyrinthitis should not be subjected to operation on the labyrinth* for two quite obvious reasons. First, such cases rarely, if ever, lead to intracranial complication, and, second, a partial or even a complete return of function takes place in many of these cases, whereas an operation on the labyrinth will result in complete and permanent destruction of function. Another conclusion which becomes quite apparent is that all cases where there is a complete suppression of function are not cases of diffuse suppuration of the labyrinth. Many of these are apparently cases of severe serous or rather, sero-purulent labyrinthitis. In not a few cases there will be later at least a partial return of function. The danger of an intracranial complication in such cases is so remote that an operation on the labyrinth is not justified. Finally, this conclusion is quite apparent: That only those cases of diffuse labyrinthitis require an operation on the labyrinth where there is complete suppression of function, and of these cases only those require a labyrinth operation where the clinical indications for an operation on the mastoid are present, and where the drainage of the labyrinth is made a part of the mastoid operation; or where, in the absence of such symptoms of mastoiditis, there develop symptoms indicating the possible beginning of an intracranial complication. In our opinion, these conclusions regarding the indications for operation on the labyrinth apply as well to the cases of acute labyrinthitis occurring as a complication of chronic otitis media as they do to cases occurring as a complication of acute otitis media. Cases of chronic purulent otitis media in which the recognized clinical indications for the radical mastoid operation exist, should have the labyrinth drained as a part of the mastoid operation, provided there is evidence of a diffuse labyrinthitis with complete loss of function.

Labyrinth infection secondary to meningitis.—Labyrinthitis occurring as a complication of cerebrospinal meningitis differs in no important respect in its clinical symptoms from the same process occurring as a complication of otitis media. When both ears are simultaneously involved, as is often the case in meningitis, the symptoms of disturbed equilibrium may be largely wanting. Here the simultaneous bilateral loss of labyrinth tonus does not give rise to the unbalance which is the conspicuous symptom of unilateral acute labyrinthitis. The vertigo and nystagmus, as well as nausea and vomiting, are absent. The patient suffers from a marked muscular weakness as the result of the sudden loss of labyrinth tonus. It is usually some weeks before compensation develops sufficiently to take the place of the lost labyrinth tonus. The loss of function in the internal ear

may not be complete in these cases. Islands of hearing, so-called "tone islands," are not infrequently discovered in the midst of the tone scale, if an examination is made of the hearing.

Primary disease of the labyrinth.—Cases of primary disease of the labyrinth present many points of interest. Accurate knowledge regarding these conditions has been hard to obtain, because of the difficulties which surround the study of their pathology. Certain facts have, however, become established, as much perhaps through the study of the clinical symptoms as by actual post mortem examinations. That syphilis plays an important part in the occurrence of primary labyrinthitis is well known. Involvement of the internal ear is frequently an early symptom of secondary lues, where its presence is shown by the development of a continuous high-pitched tinnitus and a circumscribed defect in the hearing restricted to the upper part of the tone scale. A more or less marked shortening of the bone conduction is also characteristic of this trouble. An involvement of the vestibular apparatus in such a way as to cause vertigo is not so common, but does occur. Degeneration in the labyrinth is not uncommon in tertiary lues. The process is often insidious in its development, where it causes only the usual symptoms of tinnitus and a gradual development of nerve deafness, without ever giving rise to vertigo. Fulminating cases of labyrinthitis in tertiary lues are, however, not so uncommon. Here, in the presence of a high-pitched tinnitus, there develops a rapidly increasing nerve deafness. These cases are not infrequently associated with severe attacks of vertigo, which always indicates a rapid destruction of the function of the vestibular apparatus. Hereditary lues is also prone to involve the internal ear, producing tinnitus and nerve deafness, with or without attacks of vertigo. The presence of vertigo is dependent again not so much on whether the vestibular apparatus is involved but rather on the occurrence of acute exacerbations of a degenerative process involving the vestibular mechanism.

The question of *labyrinth disturbance as a result of hemorrhage into the internal ear* is a very interesting one. Ménière attributed the sudden attacks of vertigo associated with tinnitus and deafness in the eleven cases reported by him to hemorrhages into the labyrinth. It seems doubtful now whether these symptoms in any of the cases reported by Ménière were the result of hemorrhage into the labyrinth. No one can doubt that hemorrhage into the internal ear will produce the characteristic Ménière symptom-complex, but it seems extremely doubtful whether such hemorrhages are frequently the cause of these symptoms. Hemorrhages into the labyrinth are known to occur especially in such constitutional diseases as pernicious anemia, leukemia, and purpura. An embolus lodging in the labyrinthine artery will also give rise to the characteristic Ménière sym-

tom-complex. Except, however, for air emboli in caisson workers, this is even more rare than an actual hemorrhage.

Neuritis of the eighth nerve is a very frequent cause of primary involvement of the labyrinth. This neuritis may be the result of poisoning from drugs, such as quinine, tobacco, or alcohol. The disturbance in such cases is usually temporary and disappears quickly after the drug is discontinued. Neuritis, as the result of the toxic action of infectious fevers, is met with especially in measles, mumps, scarlatina, diphtheria, typhoid fever, and florid tuberculosis. In these cases the process may involve either the cochlear or vestibular nerves alone, but frequently it involves both of these nerves. Where the cochlear nerve alone is affected, a high-pitched tinnitus with typical nerve deafness are the only symptoms. Whenever the vestibular nerve is affected there will be attacks of vertigo associated with nystagmus, provided the process is associated with acute exacerbations. On the other hand, an insidious involvement may proceed without once causing attacks of vertigo. Involvement of the vestibular nerve in these cases can be readily detected, as a rule, by means of the turning tests or by caloric stimulation of the semicircular canals—douching with cold or hot water.

The most frequent condition producing nerve deafness and vertigo is a *primary degenerative process involving, as a rule, both the cochlear and vestibular nerves*, which develops independent of either syphilis or the infectious fevers. The progress of the condition is often quite insidious, producing only tinnitus and deafness, but it is not uncommon for it to be punctuated by distinct attacks of acute exacerbations. These acute attacks always give rise to the typical Ménière symptom-complex, provided both parts of the eighth nerve are involved. The severity of the symptoms varies greatly, not only in different cases but in different attacks of the same case. Occasionally a single attack destroys permanently the entire function of the labyrinth. In these cases there can be, of course, but one attack, unless it should happen that the opposite ear becomes later involved. In cases where the function of the labyrinth is only partially lost there is, as a rule, a series of these attacks, extending occasionally over a period of several years and occurring at irregular intervals.

It is these cases of primary involvement of the labyrinth, with sudden acute exacerbations producing the characteristic *Ménière symptom-complex*, that have been reported from time to time as cases of Ménière's disease, and it was assumed that only hemorrhage into the labyrinth could account for the attacks. In the milder attacks, especially where the symptoms were quite transitory, it was often assumed that they were the result of an angio-neurosis. As a matter of fact, it is not possible to draw a sharp distinction between the

more severe attacks, where a hemorrhage into the labyrinth could account for the symptoms, and the milder cases where, from the transitory character of the symptoms, an actual hemorrhage into the labyrinth could be positively excluded.

These cases of *primary involvement of the internal ear not associated either with lues or the infectious fevers*, may be the result of a degenerative neuritis of the eighth nerve, produced by such foci of infection as exist so frequently in the faucial tonsils and about the teeth. This origin of the labyrinth symptoms accounts readily for the sudden attacks which result in the production of vertigo. Sudden acute exacerbation is a characteristic symptom observed in the course of neuritis occurring elsewhere as the result of focal infection. Each acute attack registers the liberation of a fresh shower of bacterial emboli into the circulation, which lodge now in this nerve, now in that, as the result of some not as yet understood selective action. For obvious reasons, it will be more difficult than is the case in involvement of other nerves to demonstrate therapeutically, that is, by the removal of the primary focus, that the degeneration of the eighth nerve is the result of a focal infection. It is only the acute exacerbations which produce the characteristic Ménière symptom-complex in these cases, and a single attack may result in a permanent destruction of function of the eighth nerve, when, of course, no subsequent attacks are possible. It is, moreover, a well-recognized clinical fact that these attacks of vertigo disappear spontaneously. This may occur after only one or two attacks which leave only a partial loss of function, while in other cases the disappearance of the attacks takes place only after the complete destruction of the function of the labyrinth, as can be demonstrated by the turning tests or by caloric stimulation.

Examination of the internal ear.—It is not our intention to discuss here the details of an otologic examination. Our aim is rather to point out briefly the more important methods employed in making an examination of the function of hearing, as well as of the function of the vestibular mechanism, with the object in view of determining on the one hand whether the disturbance in hearing is due to an obstruction in the sound-conducting mechanism in the middle ear or to a disease involving the sound-perceiving apparatus in the internal ear, and, on the other hand, to determine whether the vestibular mechanism is functioning normally.

Cochlea.—The *diagnosis* of a disease of the sound-perceiving mechanism in the cochlea from a defect in hearing which is the result of disease interfering with the conduction of sound impulses from the outer air into the labyrinth can be made only by the functional examination of the hearing. The presence of a disease of the con-

ducting mechanism in the middle ear gives no definite assurance that the chief cause for an existing defect in the hearing may not be from internal ear disturbances. It is not uncommon, for example, to examine cases where the presence of a marked retraction of the membrana tympani points conclusively to a long-continued tubal occlusion, but where the functional examination discovers no defect in the hearing persisting from the exhausted tubal catarrh. Cases, moreover, present themselves not infrequently where an exhausted chronic suppurative otitis media has left a complete destruction of the membrana tympani with the loss of the malleus and incus, but where the movement of the stapes has remained unimpaired and where only a very slight defect in the hearing is discovered. On the other hand, the presence of an entirely normal drum-membrane can not be accepted as evidence that an existing defect in the hearing is due to a disease of the internal ear. The most profound obstruction of the sound conducting mechanism results from primary bony fixation of the stapes with the drum membrane quite normal.

The *first principle* to bear in mind in making a diagnosis of internal ear disease is that a profound defect in the hearing can arise only from a disturbance in the sound-perceiving mechanism. Complete occlusion of the external meatus or fixation of the sound-conducting mechanism in the middle ear either from adhesions in the middle ear from catarrh or as the result of acute or chronic suppurative disease of the middle ear, or even from complete bony fixation of the stapes, can result only in a moderate depression of the hearing function, so long as the sound-perceiving mechanism in the labyrinth remains unimpaired. As a matter of fact, however, a secondary nerve degeneration does take place sooner or later in most cases where there has been a long-standing fixation of the sound-conducting mechanism from any cause.

The *second principle* in diagnosing a defect in hearing due to internal ear disease is that this defect almost without exception begins in the upper part of the tone scale. Not until the defect becomes very extensive is there any appreciable depression of hearing for the lower tones. This is in striking contrast to the defects in hearing occasioned by an obstruction in the sound-conducting mechanism, where the defect is always limited to the lower part of the tone scale. The presence in such cases of a coexisting defect in the upper part of the tone scale always indicates a secondary degenerative process in the labyrinth. The use of the whispered voice is a simple and often quite satisfactory method of making this diagnosis. For example, when a patient discloses a distinct difficulty in hearing a number like 66 but does hear quite readily numbers like 18 and 23, the conclusion is quite definite that the defect in hearing is

the result of disease of the sound-perceiving mechanism, and vice versa. The examination is made more complete and the diagnosis more accurate by testing the upper part of the tone scale with a Galton whistle, or the c^4 tuning fork vibrating 2,048 times per second, and by testing the lower tone registered by means of a low-tuning fork, as for example C vibrating 64 times per second.

A number of *tuning-fork* tests may be used by otologists in differentiating between defects in hearing caused by disease of the sound-conducting apparatus and defects due to disease of the sound-perceiving mechanism. These tests are all dependent on two well-recognized principles in bone conduction. One is that when there is a defect due entirely to an obstruction in the conducting mechanism, the hearing by bone conduction—that is, when a fork is placed on the skull—is better than in a normal case, and the more profound the defect in hearing the better will be the ability to hear by bone conduction. The second principle is that when the defect in hearing is due entirely to an involvement of the internal ear, there will be a distinct lowering of perception by bone conduction. In cases where the nerve defect is at all severe, the patient will not hear the tuning fork at all when placed on the skull. Such a loss of bone conduction presupposes, of course, that both ears are involved, since it is not possible to examine each ear separately by bone conduction. In testing for bone conduction, neither the higher nor the lower forks are suitable. The best fork for this purpose is the "A" or the middle "*c*." The "A" fork (Bezold) is especially suitable for testing duration of bone conduction, because it dies out so slowly. The lateralization of the hearing by bone conduction for the tuning fork to the affected ear, when the defect is the result of obstruction in the sound-conducting mechanism, or vice versa when the defect is in the labyrinth, is another test used in diagnosing the cause of deafness. Comparing the duration of hearing for the tuning fork by bone and air conduction is another test used in differentiating nerve deafness from conduction deafness. The intelligent interpretation of this test requires a great deal of experience; it is a test, therefore, that can be used only by the trained otologist.

The diagnosis of a complete unilateral suppression of hearing is not a simple matter, because it is not possible to exclude the opposite ear by closing the external meatus as in testing for bone conduction. The production of a loud noise in the opposite ear either by using a specially devised noise apparatus or by inserting the handle of a sounding tuning fork into the external meatus efficiently excludes this ear, but this procedure also lessens the ability to hear in the affected ear, and when the margin of hearing is only slight it will not be possible to discover this margin while making a noise in the oppo-

site ear. As a matter of fact, an experienced otologist finds little difficulty in diagnosing a complete or practically complete suppression of hearing in one ear without the use of the noise apparatus, but for the inexperienced the surest way of making this diagnosis is by the use of such a device.

Semicircular canals.—As the physiology of the semicircular canals is much simpler than that of the cochlea, so the *functional examination of the vestibular apparatus* is a *much more simple* procedure than that of hearing. The disturbance in equilibrium arising from a recent disturbance in the function of the semicircular canals is quite characteristic. The patient when attempting to stand with feet together and eyes closed tends to fall toward the affected side. This will be more readily discovered when the patient attempts to stand on one foot. If he attempts to walk toward a known point, eyes closed, he will deviate toward the affected side, and when he attempts to touch a known point with the index finger, swinging the arm freely upward from the shoulder, he will past-point, also toward the affected side. This past-pointing will be observed even in cases where the patient is free from any sensation of vertigo or of falling, and under these circumstances is clearly due to the same influence which causes the patient to deviate to the affected side, namely, to the tonus impulses from the normal labyrinth acting without the restraint of the opposing impulses from the affected labyrinth. During the early stages after a disturbance of the semicircular canals, there will be present a spontaneous nystagmus in which the eyes are drawn slowly toward the affected side, only to move quickly back again toward the normal side. In the severe cases, this spontaneous nystagmus will be present no matter what the position of the eyes, but will become more apparent when the eyes are directed toward the normal side. In other cases the spontaneous nystagmus disappears entirely when the eyes are turned strongly toward the affected side, or is observed only when they are turned toward the normal side.

The tests.—The examination of the semicircular canals for practical purposes is made by causing a flow of endolymph in one or more of the canals. This is accomplished either by turning a patient in a rotating chair, which, of course, affects both labyrinths simultaneously, or by cooling or warming the outer wall of the labyrinth by injecting cold or warm water in the external canal. By the latter method it is possible to examine each labyrinth separately.

Turning test.—This test is made by using a rotating chair. The patient sits in the upright position with the head tipped slightly forward, so that a line drawn through the middle of the eye and the external meatus is parallel with the floor, which brings the horizontal canals exactly in the plane of rotation. The patient is turned slowly in one direction, making 10 rotations in 20 seconds. On

stopping rotation, the character and duration of the nystagmus is noted. This nystagmus will have the quick component directed away from the direction of rotation. In a normal case the nystagmus will last approximately 26 seconds. The patient is then rotated toward the opposite side in exactly the same way. As will be seen from examination of Figure 61, the nystagmus resulting from rotating the patient to the right will be caused chiefly from impulses emanating from the left labyrinth, and the quick component of the nystagmus will be toward the left, because on stopping rotation it will be the more sensitive group of hair cells in the crista of the left horizontal canal—those on the side of the crista toward the canal—which are

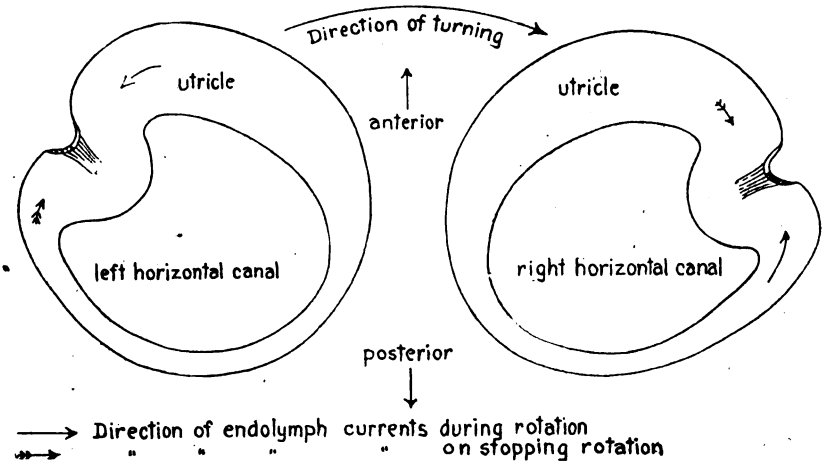

FIG. 61.—Schematic representation of the two horizontal semicircular canals to show the movement of endolymph during and after rotation. Note that the current of endolymph after rotation to the right will stimulate the more sensitive hair-cells in the crista of the left horizontal canal and the less sensitive group in the crista of the right canal. Since the slow component of the resulting nystagmus is always in the direction of the endolymph current, it is evident that the impulses from both canals tend to produce a nystagmus with the quick component to the left, the stronger impulses emanating from the left canal. In the case of the destruction of one labyrinth, the nystagmus after rotation will be stronger when the patient is being rotated toward the affected side. (Shambaugh.)

being stimulated by the endolymph current. When the patient is rotated toward the left, the nystagmus resulting after stopping turning will be caused chiefly by impulses emanating from the right labyrinth, and the quick component will be toward the right. When one labyrinth is destroyed the nystagmus resulting from rotation toward the opposite side will be naturally much less than when the patient is rotated toward the affected side. The reason why there results any nystagmus at all in such a case after rotation toward the normal side is because the current of endolymph set up by rotation in the normal labyrinth stimulates in this canal the less sensitive group of hair cells—that is, the group on the vestibular side of this crista—and, as

is pointed out under "Physiology of the Semicircular Canals," these are the cells which, when stimulated, produce movements toward the opposite side. The eyes, therefore, are drawn toward the normal side, with the quick component of the nystagmus directed toward the affected side. Only in cases of long-standing destruction of the labyrinth will the reaction be the same after rotation in either direction. The equalization of the response from the stimulation of the hair cells on the two sides of the same crista is apparently the result of nature's efforts at reestablishing the equilibrium of tonus impulses after one labyrinth has been destroyed.

Only the canals which are in the plane of rotation can be stimulated by the rotation tests. With the head upright, that is, tipped forward 30°, only the horizontal canals are stimulated. It is possible by tipping the head forward 120° or backward 60° to bring the vertical canals in the plane of rotation. The nystagmus resulting after rotation in this position will be in the plane of the vertical canals, that is, rotary in character. If the head is raised to the upright position after rotation the patient *falls* to the side toward which he was being rotated.

Caloric stimulation.—By this test it is possible to test separately not only each labyrinth but both the superior and horizontal canals. The posterior canal is located too far away from the middle ear to be influenced by caloric stimulation. The caloric stimulation of a semicircular canal is the result of an endolymph current set up by cooling or warming the external wall of the labyrinth. It is evident, therefore, that only the canals which are in the vertical plane can be affected by this test. With the head in the upright position the superior canal will be stimulated, and with the head tipped forward so as to place this canal in the horizontal plane the horizontal canal will be stimulated, because now this canal is in the vertical plane.

The test is usually carried out by using cold water, 68° F., which is allowed to run in a steady stream into the external meatus of the ear to be examined. When there is a perforation of the membrana tympani left from an exhausted chronic suppurative otitis media, the test should be carried out by means of a compressed air current, as the water is pretty sure to bring on an acute exacerbation of the exhausted otitis media. With the head in the upright position, the cold water in the external meatus will cool the external crus of the superior canal, causing a flow of endolymph from above down, that is, from the canal toward the vestibule, thus stimulating the hair cells on the side of the crista toward the canal. The result will be a nystagmus in the plane of this canal, that is, a rotatory nystagmus. The slow component will be in the direction of the endolymph cur-

rent, that is, toward the side being stimulated, with the quick component directed toward the opposite side. Tipping the head 120° forward places the superior canal in the horizontal plane, causing an immediate cessation in the flow of the endolymph and a cessation of the rotatory nystagmus. This position of the head places the horizontal canal in the vertical plane and causes a movement of the endolymph from the canal toward the vestibule, thus stimulating the hair cells on the side of the crista toward the canal. This, as is pointed out under "Physiology of the Semicircular Canals," causes a horizontal nystagmus with the quick component directed toward the same side. If warm water is used the flow of endolymph will be exactly opposite from that caused by cold water, and the resulting nystagmus will be reversed.

Pointing tests.—These tests are carried out either without stimulation of the semicircular canals in order to ascertain the spontaneous pointing reactions, or after stimulation of the canals. In applying the pointing tests the patient sits in a chair with eyes closed. He extends his arm horizontally in front of him and with his index finger touches the examiner's finger, which is held stationary at a certain point. He is then instructed to swing his arm freely upward without bending the elbow, and told to again touch the finger of the examiner. The normal individual experiences no difficulty in retouching the examiner's finger with his eyes closed. Deviations from the normal in the spontaneous pointing reactions are found in cases of labyrinth disease producing disturbance of equilibrium, and are especially marked where the disturbance is causing a spontaneous nystagmus. In these cases the patient will past-point to the side of the slow component of the nystagmus. Spontaneous past-pointing is found also in cases where the peripheral mechanism in the labyrinth is intact, but where a central disturbance interferes with the central course of the vestibular fibers.

After stimulation of a functioning labyrinth the patient past-points in the direction of the slow component of the resulting nystagmus. That the past-pointing may be in part dependent on conscious cerebral impulses is suggested by the fact that after rotation with the head tipped back, the past-pointing will be in the direction of the rotation, although the resulting nystagmus has its slow component directed to the opposite side. In applying the pointing test after rotation, the patient is turned ten times in ten seconds, eyes closed and head erect. If the labyrinths are functioning and there is no central interference with the passage of impulses along the central pathway of the vestibular nerve, the patient will not be able to touch the examiner's finger, but will past-point toward the side to which he had been rotated. After caloric stimulation the patient

with functioning semicircular canals is unable to again touch the examiner's finger, but past-points toward the side to which the slow component of the resulting nystagmus is directed. For example, after cold stimulation of the right ear with the head upright, the patient will past-point to the right, and the quick component of the nystagmus will be to the left.

These are the normal pointing reactions, and are the responses elicited even where there is some depression of the function of the labyrinth, but where this depression is not sufficient to inhibit completely all response from stimulation of the semi-circular canal. In cases where an intracranial disease interferes with the transference of impulses from the semi-circular canals, the normal past-pointing after stimulation is interfered with. In some cases the past-pointing may be absent entirely, while the nystagmus resulting from the stimulation may be normal. In other cases the past-pointing may be reversed. For example, after rotation to the right, the patient with a central disturbance may either fail altogether to past-point or he may past-point to the left instead of to the right. In the same way after caloric stimulation in cases of an intracranial disorder, the past-pointing may be either absent or atypical. In some cases, the past-pointing after rotation may be interfered with, while the reaction after caloric stimulation remains normal, or vice versa. Such responses are always significant of a central disturbance, since they can be occasioned only by a lesion which interferes with the transmission of impulses from one canal, while the function of the fibers from the other canal remains undisturbed.

The ear and the neurologist.[1]—The intimate relation of the ear to the central nervous system makes the study of neuro-otology of especial interest to the neurologist. The value of an eye-examination in neurologic cases is now universally recognized. The study of the eye-grounds, the field of vision, pupillary reaction to light and accommodation and the degree of function of the eye-muscles, furnishes valuable information to the neurologist. Based on the opinion of the neurologists and ophthalmologists most acquainted with these ear-tests, it would seem safe to assert that of the two methods of approach, very much more definite information can, as a rule, be had from the examination of the vestibular apparatus than from an eye-examination. The value of ear-examination in neurologic cases is recognized to the extent that no examination of a neurologic case is considered complete without a report from the otologist as to the condition of the vestibular apparatus.

The neurologist is not only concerned with the general problems of equilibration which have already been presented, but, in addition, he

[1] From Equilibrium and Vertigo, by Isaac H. Jones, M. D. J. B. Lippincott Co.

has, in these ear-examinations, a very definite help in many perplexing diagnoses. The ear-tests are of particular value in making a differential diagnosis between labyrinth and intracranial lesions and in furnishing additional data in intracranial localization.

It is well known that nystagmus and vertigo, with loss of equilibration, associated perhaps with nausea and vomiting, may be produced either by a disturbance of the internal ear or by an intracranial lesion. In many instances the symptoms of internal ear disturbance and of a cerebellar lesion are identical. It is in such a differentiation that the ear-tests are often invaluable. It not rarely happens that a careful neurologic study indicates a lesion of the cerebellum, whereas the ear-examination, by giving additional data to the neurologist, demonstrates conclusively that he is dealing with a lesion of the labyrinth.

A differential diagnosis between peripheral and central lesions by means of the ear-tests depends on certain general principles. A peripheral lesion of the labyrinth or eighth nerve is suggested by the following:

(1) An impairment of the function of both the cochlear and kinetic-static labyrinth. If, for example, the hearing tests show cochlear deafness and the tests of the semicircular canals show that their function is also impaired, it immediately becomes probable that we are dealing with an end-organ lesion.

(2) The history of presence of tinnitus; the absence of tinnitus does not necessarily indicate that the end-organ is not involved, but its presence is very suggestive of labyrinth involvement.

(3) Proportionate impairment of the responses from the horizontal canal and of the responses from the vertical canals. If, for example, the tests show that the horizontal canal retains only one-half of its normal function and that the vertical canals similarly retain only one-half of their normal function, a lesion of the end-organ itself is suggested.

(4) Proportionate impairment of both nystagmus and vertigo. If the horizontal canal produces one-third of the normal nystagmus and one-third of the normal vertigo, it is suggested that the lesion is in the horizontal canal itself or in the fibers from the canal within the eighth nerve; if, in addition, the vertical canals produce one-third of the normal nystagmus and one-third of the normal vertigo, an end-organ lesion is strongly suggested.

In a word, it is the "proportionate impairment" of responses that speaks for a peripheral lesion.

A central lesion is suggested by the following:

(1) A normal cochlea, but impaired or non-responsive semicircular canals.

(2) Normal responses from the horizontal canal, but absent responses from the vertical canals.

(3) Normal responses from the vertical canals, but impaired responses from the horizontal canal.

(4) Normal vertigo, but impaired nystagmus from the horizontal canal.

(5) Normal nystagmus, but impaired vertigo from the horizontal canal.

(6) Normal vertigo, but impaired nystagmus from the vertical canals.

(7) Normal nystagmus, but impaired vertigo from the vertical canals.

(8) Normal vertigo and normal nystagmus from any semicircular canal, but impaired past-pointing in any direction of any one extremity.

(9) Normal vertigo and normal nystagmus from any semicircular canal, but an impairment or absence of the normal falling.

(10) Spontaneous vertical nystagmus is pathognomonic of a central lesion and is indicative of involvement of the brain-stem caused either by infiltration or pressure. A lesion of the labyrinth may produce many forms of spontaneous nystagmus—horizontal, rotary, oblique, or a mixed nystagmus of the various types; but an ear lesion can never produce a spontaneous vertical nystagmus, either upward or downward.

(11) If there exists a spontaneous nystagmus to the right and non-responsive semicircular canals of the right ear, an intracranial lesion is suggested. The non-responsive labyrinth, if the labyrinth itself alone were responsible, would produce a nystagmus to the left.

(12) A spontaneous nystagmus of increasing intensity or of long duration is indicative of a central lesion. A spontaneous nystagmus due to a lesion of the labyrinth shows its greatest intensity at the onset of the disease, becomes less and less marked and disappears after a few days.

(13) If a stimulation of any semicircular canal produces a "perverted" or "inverse" nystagmus, it is pathognomonic of a central lesion and is indicative of brain-stem involvement. Such phenomena as the following are frequently seen: Douching the right ear with cold water with the head back, stimulating the right horizontal canal, should produce a pure horizontal nystagmus to the left. If on such stimulation there occurs a vertical nystagmus upward or downward, a rotary, oblique, or mixed nystagmus, it may be spoken of as "perverted." If, instead of a horizontal nystagmus to the left, there is produced a pure horizontal nystagmus to the right, it may be termed an "inverse" nystagmus. Neither a perverted nor an inverse nystagmus can possibly be produced by a lesion of the labyrinth or

eighth nerve; a peripheral lesion produces a poor nystagmus or no nystagmus at all, but an absolutely false response of necessity demonstrates a central lesion.

(14) If ear-stimulation produces a conjugate deviation of the eyes instead of a nystagmus, it is pathognomonic of a central lesion.

The above outline indicates how additional data may be furnished to the neurologist by the ear-tests in determining whether he is dealing with a lesion of the internal ear or of the brain-stem or cerebellum. In the broader field of intracranial localization, examination of the ear and of the vestibular apparatus is also of distinct value. The particular feature of the ear-examination is that the aurist sends in a stimulus to the brain-centers, and then notes the responses of different parts of the body to this stimulus. For example, by stimulation of the ear there results a nystagmus in a given direction, a pointing of the extremities to the right or to the left, as the case may be, and a falling to the right, to the left, forward or backward, as the case may be. Now, if the ear and these nerve paths from the ear are intact, all the normal responses will appear; if there is a failure of all or any of the responses it is positive evidence of an interruption along that particular path or paths that fail to bring about these responses.

In order to utilize the knowledge obtained from these tests it is essential to have in mind the various pathways constituting the vestibular apparatus. It may be stated at this point in brief that the pathways from the horizontal semicircular canal are different after entering the brain-stem from those from the vertical canals; furthermore that each set of tracts divides into two separate pathways; one pathway, the vestibulo-ocular tract, is responsible for the eye movement, and the other pathway, the vestibulo-cerebello-cerebral tract, conveys the impulses from the ear to the cerebral cortex, producing vertigo. If the horizontal canal fails to produce both nystagmus and vertigo the lesion indicated is at a point before the division of the horizontal canal fibers into their two separate pathways. Further, if the horizontal canal produces normal vertigo but no nystagmus, the lesion indicated is of the vestibulo-ocular tract at a point beyond the point of division into the two paths. If the horizontal canal produces normal nystagmus but no vertigo, the lesion indicated is at a point along the vestibulo-cerebello-cerebral path beyond the point of division into the two pathways. Similarly if the vertical canals produce normal vertigo but no nystagmus the lesion indicated is in the vestibulo-ocular tract at a point beyond the division into the two paths. If the vertical canals produce normal nystagmus but no vertigo, the lesion indicated is at a point along the vestibulo-cerebello-cerebral path beyond the point of division into

the two pathways. The ear tests have proved themselves surprisingly helpful in locating lesions in the cerebello-pontile angle, medulla oblongata, pons, cerebellar peduncles, cerebellum and various portions of the cerebrum, including the parietal lobe, the temporal lobe, and the occipital lobe.

In order to obtain reliable data from an ear-examination, it is essential that the technic of examination should be accurate and painstaking; since it is primarily an ear-examination, the otologist is peculiarly fitted to carry out such examinations. Although one purpose of this book is to furnish a practical guide for the otologist in undertaking the examination of patients, it is also offered to the neurologist so that he, on his part, may become familiar with the ear aspects of the work, in order to realize the significance of the reactions as reported to him. The ear-examination is obviously not for the purpose of making a neurologic diagnosis; it merely gives additional information by a series of refined experiments to the other methods at the command of the neurologist. To be sure there are many cases in which the neurologist, without the aid of the ear, eye, blood, or other examinations, finds no difficulty in arriving at a satisfactory diagnosis. In these cases, however, it is of course useful to have the additional evidence from the ear-tests corroborating his neurologic and other findings. In addition, it not infrequently happens, in obscure cases, or in cases in which the neurologic data are meager, that the ear-tests may be the only means of furnishing information upon which a diagnosis can be made. For example, an apparently strong, vigorous man, complaining only of headache, showed on examination that both internal ears were normal and also that both eighth nerves were normal, and yet the vertical semicircular canals of both ears, when stimulated, failed to produce any responses whatever. The horizontal canals produced normal nystagmus but no vertigo. As the labyrinths and eighth nerves in this case were unquestionably normal, the nonappearance of the normal responses to stimulation could be accounted for only by an interference with the fibers from the labyrinth within the brain-stem. This particular phenomenon-complex indicates pressure within the fourth ventricle. This conclusion was recorded with considerable misgiving because a lesion within the fourth ventricle appeared ridiculous in view of the man's apparent health. That night the patient was rushed to the hospital unconscious. The next day he regained consciousness but complained of agonizing headache. Examination by a number of internists and neurologists failed to give any clue of an organic lesion anywhere, and the diagnosis of hysteria was made. Autopsy three days later showed abscess in the fourth ventricle. It is in such cases

as this that the ear examination is of the utmost importance, as it gives data absolutely unobtainable by the neurologic tests.

The ear and the surgeon.—The diagnosis of the precise location of lesions within the cranium is probably the most difficult task with which the surgeon is confronted. Fully realizing all the difficulties in these cases, he utilizes every modern aid in diagnosis—the laboratory, the X-ray, or any other instruments of precision that may be available. For many years the ophthalmologist has been of great help to the surgeon in his intracranial cases, and no one would think of operating on such a case without first having the ophthalmologist's report as to his findings. It is shown elsewhere how the ear-tests may furnish data of value in the accurate localization of lesions which involve any of the vestibular pathways, as well as in differentiating between labyrinth and intracranial lesions. A point worthy of special emphasis is that, with the aid of these tests, he is frequently enabled to determine whether a lesion is operable or inoperable. Many lesions of the medulla oblongata, pons, or cerebellar peduncles, which are inoperable by the very nature of their location, will show very pronounced cerebellar symptoms, and not infrequently the cerebellum is explored, in such cases, in the hope of finding a tumor near the cortex, and removing it. For such a differentiation, in many cases, no method can equal the accuracy of the Barany tests. If, after turning and douching, there appears a normal past-pointing of both extremities in both directions, it may safely be assumed that the cerebellum itself is intact. This can not be regarded as absolutely final, but is much more definite than any other known method for determining the integrity of the cerebellum. It is perfectly conceivable that a case showing almost normal past-pointing may apparently reveal a lesion when a cerebellar decompression is done. We have seen two such cases. Here, however, it should be explained that in both these instances the lesion proved to be a cyst which grew from the brain-stem and extended backwards between the cerebellar hemispheres, without, however, destroying the substance of the cerebellum itself. Such cases give definite neurologic evidence of a cerebellar lesion, and at the time of operation, such a lesion is apparently discovered. There may have been present asynergy, manifested by hypermetry, adiodokokinesis and tremor, yet these symptoms or phenomena were produced by involvment of the fibers on their way to the cerebellum, or by pressure upon the cerebellum itself. If, then, the ear-tests demonstrate normal past-pointing, it is strong evidence that the cerebellum itself is not involved.

Tumors in the cerebello-pontile angle either originate from one of the cranial nerves in the angle, usually the eighth nerve, or have

invaded the angle secondarily from the cerebellum or brain-stem. These tumors invading the angle from other adjacent structures also usually involve the eighth nerve. (Fig. 62.) Obviously then a careful study of the various portions of the eighth nerve gives direct insight as to conditions in the cerebello-pontile angle. In addition, in our experience the ear-tests have usually demonstrated the two following phenomena, in cases of cerebello-pontile angle growth:

(1) An absence of all responses from the vertical canals of the ear opposite the side of the lesion. Given a tumor in the right cerebello-pontile angle, the usual findings are as follows: The right ear gives no responses—the cochlea shows absence of all function and the horizontal and vertical canals fail to produce any nystagmus, vertigo, past-pointing, or falling. The left ear shows unimpaired hearing, and the left horizontal canal does produce nystagmus, vertigo, past-pointing, and falling. The left vertical canals, however, fail to produce any responses, because the vertical canal fibers are most probably impaired because of pressure upon the pons by the tumor in the angle.

(2) "*Crossed past-pointing.*"—This phenomenon consists of persistent past-pointing of both upper extremities either outward or inward, regardless of the type of ear-stimulation employed.

If the ear-stimulation produces normal nystagmus, vertigo, past-pointing, and falling, the Barany tests are of unquestioned value in eliminating lesions in the posterior fossa and brain-stem. If they rendered no other service than this it would be sufficient to hail them as a distinct contribution in the diagnosis of intracranial lesions. In addition these tests may prove helpful to the surgeon in preventing unnecessary operations. One case, given in detail in Chapter XXIII, page 308, case No. 2, "Equilibrium and Vertigo," is of interest in this connection: A woman appeared to have a tumor of the right cerebellar hemisphere. This diagnosis was confirmed by neurologic consultants, and the X-ray report stated that it was a cyst in the right cerebellar hemisphere. The ear-tests suggested that the cerebellum was uninvolved; operation was delayed on this account and eventually was found to be unnecessary, as the patient steadily improved and was discharged from the hospital apparently well, and for years has had no recurrence of her symptoms. Her cerebellar symptoms evidently were due to a disturbance in the internal ear.

No operation upon the brain should be undertaken without giving the patient the benefit of an ear-examination.

Glossopharyngeal.—The glossopharyngeal nerve supplies motor and sensory fibers to the pharynx, and taste fibers to the posterior portion of the tongue. Division of the nerve will cause difficulty in swallowing, diminution or loss of sensation over the pharynx.

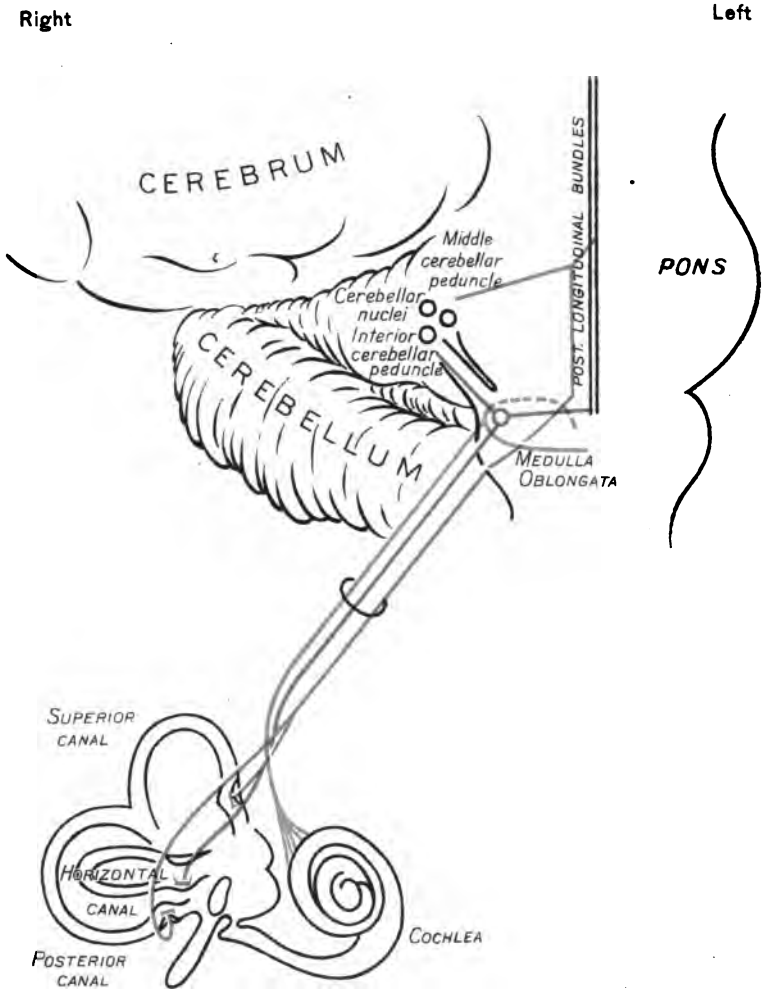

Fig. 62.

Pathways of fibres from cochlea, horizontal canal and vertical canals.

Viewed from in Front.

Vagus.—The vagus nerve arises from the medulla, leaves the skull through the jugular foramen, and passes down the neck with the great vessels. It supplies inhibitory fibers to the heart, fibers to bronchi, lungs, esophogus, stomach, and intestines, fibers to the soft palate, pharynx, and larynx. The recurrent laryngeal branch supplies all the muscles of the larynx except the cricothyroid.

Injury to the nerve may result in hoarseness and weakness of the soft palate. The hoarseness results from involvement of the vocal cord on the affected side; on laryngological examination no movement of the cord is seen. To test the movements of the soft palate,

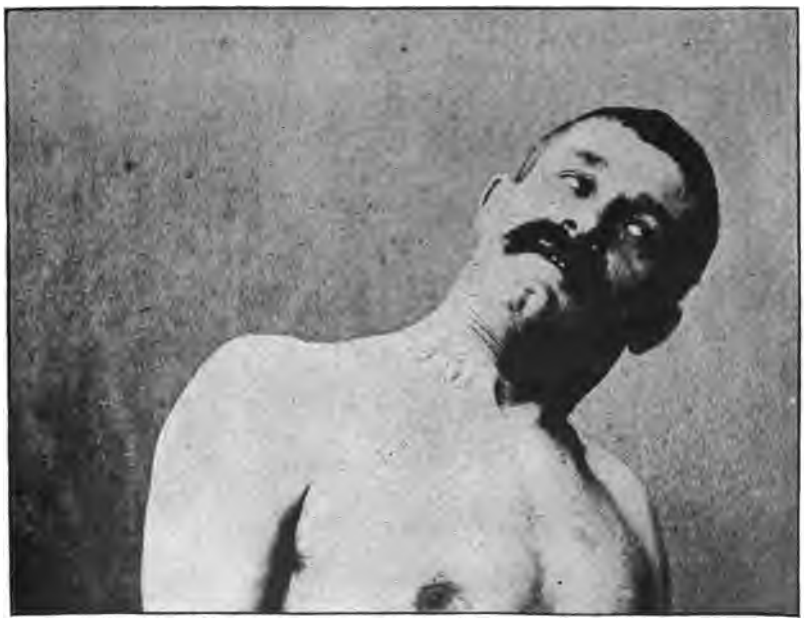

FIG. 63.—Torticollis from irritation of right spinal accessory nerve. Illustration shows deviation of head and contraction of muscles of the back of the neck. (Weisenburg.)

the patient is made to say "Ah." The palate in moving up is deflected toward the sound side.

Spinal accessory.—The spinal accessory arises from the medulla and cord, and leaves the skull by the jugular foramen. One part joins the vagus (vago-accessorius) and really forms an integral portion of that nerve. The other part, spinal accessory, is distributed to the sternomastoid and upper part of the trapezius. Both these muscles receive additional nerve fibers from the third and fourth cervical nerves.

The sternomastoid rotates the head to the opposite side, tipping the chin up and the vertex down. Paralysis of the muscle results

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in weakness of these movements; but all movements are still possible.

Paralysis of the upper portion of the trapezius results in displacement of the scapula down and out; elevation of the shoulder is weakened.

Hypoglossus.—The hypoglossal nerve arises from the medulla and leaves the skull through the hypoglossal canal. It is the motor nerve to the tongue.

Division of the nerve causes paralysis of the tongue on the same side. In putting out the tongue, it deviates to the affected side. Paralysis is followed by atrophy, and the reaction of degeneration.

FIG. 64.—Torticollis from irritation of right spinal accessory nerve. Illustration shows contraction of muscles of the back of the neck. (Weisenburg.)

The four nerves, IX, X, XI, XII, may be injured together by a wound at the base of the skull. This type of injury, described by Collete, results in paralysis of the same side of the tongue, vocal cord, trapezius, involvement of soft palate or pharynx, disturbance of taste and cardiac rhythm, and oculopupillary symptoms due probably to involvement of the cervical sympathetic.

True and pseudo-bulbar palsy.—*True bulbar palsy.*—In this disease there is a bilateral progressive degeneration of the motor nuclei of the medulla and pons. The disease nearly always occurs in the latter part of life and usually begins in the nuclei of the twelfth nerve and gradually extends upward involving in succession the eleventh,

ninth, seventh and fifth, and if the patient lives long enough, the ocular cranial nuclei, that is, the sixth, third, and fourth. Rarely the disease begins in the motor ocular nuclei and extends downward.

Inasmuch as each motor nerve cell represents a muscle fiber in the periphery, a gradual degeneration will be manifested in the periphery by a tremor in the muscle fiber, and as many nerve cells are implicated there will be a corresponding loss of power, atrophy, or wasting, a gradual loss of reflexes with a gradual loss of the normal electrical reactions.

This disease will therefore cause increasing tremors in the tongue, palate, and facial muscles, gradual difficulty in the use of the tongue, palate, pharynx, larynx, and facial muscles, all of which cause difficulty in talking, swallowing, eating, and chewing. Atrophy in the corresponding muscles with a gradual impairment of normal electrical reactions follows. In other words, there will be a gradual difficulty in talking, eating, swallowing, with drooling of saliva, regurgitation of food through the nose, occasional choking, wasting in all the muscles of the face, until finally the patient in the course of a year or two dies from choking. This is true bulbar palsy.

Pseudo-bulbar palsy.—In pseudo-bulbar palsy there is the same difficulty in eating, talking, swallowing, with regurgitation of food and choking, and involuntary laughing and crying, but there is an absence of certain symptoms such as fibrillary tremor, wasting, and impairment of electrical reactions, and, most important, the onset and progress are different.

It has been emphasized that in the hemiplegia due to a unilateral lesion there is an escape of the so-called bilaterally acting functions such as looking upward, moving the eyeballs, talking, eating, swallowing, breathing, laughing, and crying. It is apparent, then, that to have a disturbance of such functions there must be a bilateral lesion which will impair the fibers which control these bilaterally acting functions.

A lesion of the knee of one internal capsule which transmits the central motor cranial nerve fibers, that is, the fibers of the third, fourth, fifth, sixth, seventh, ninth, tenth, eleventh, and twelfth cranial nerves, will cause no impairment in the bilaterally acting movements which are represented in the action of the third, fourth, sixth, ninth, tenth, and eleventh cranial nerves, but will cause impairment of the unilaterally acting movements of the fifth, seventh, and twelfth cranial nerves. For example, in the fifth motor nerve which supplies the muscles of mastication, while the pterygoids and temporals do not act alone the masseters can act on one side. The lower portion of the seventh nerve, of course, can act on one side. The same thing is true of the twelfth. Therefore, a lesion of the

knee of one internal capsule will give on the opposite side impairment in the action of the masseter, the lower portion of the face, and the tongue.

A lesion, however, which involves the knee of the internal capsule on both sides will cause a greater number of symptoms, for at once there will be impairment in the action of the fifth, seventh, ninth, tenth, eleventh, and twelfth cranial nerves. That is, there will be an immediate disturbance in eating, talking, swallowing, with accompanying drooling, with the monotonous speech of bulbar palsy and regurgitation of food, etc. Usually the bilaterally acting ocular muscles escape, although sometimes they also are involved.

However, lesions of the knee of the internal capsule hardly ever occur alone, and there is nearly always an implicating of the accompanying motor fibers of the arm and leg. It is not to be inferred that the lesion must be in the vicinity of the internal capsule. The lesions may be anywhere in the brain, but they must involve the course of the *central motor cranial nerve fibers* on both sides.

The usual history of a pseudo-bulbar palsy is that of a syphilitic thrombosis of part of the middle meningeal artery which causes a hemiplegia on one side with no difficulty in eating, talking, and swallowing, followed perhaps in a year or two by another cerebral seizure with the appearance of a hemiplegia on the other side and difficulty in eating, talking, and swallowing. It is at once seen that in pseudo-bulbar palsy besides the bulbar symptoms mentioned there is an additional involvement of the limbs on both sides with increased reflexes, spastic condition, and Babinski phenomenon.

In the usual cases of pseudo-bulbar palsy there is always either involuntary laughing or crying. There are many cases in which there is no such involvement, but in the usual pseudo-bulbar patient there is a gradual emotional disturbance, usually in crying, the patient crying from the slightest provocation and while in the midst of talking. Occasionally the patient laughs only. Sometimes the patient both laughs and cries. At times, instead, there is a howling. Such involvement is dependent upon the same principle of bilaterally acting functions with bilaterally acting muscles.

Of course in this disease there are no fibrillary tremors, atrophy, or impairment of electrical reactions, because there is no degeneration of the nerve cells which supply the peripheral muscle fibers. Besides, the symptoms are not progressive, for the patient may live for years, while in true bulbar palsy death usually occurs in from two to three years.

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CHAPTER III.[1]

FRACTURES OF THE SKULL.

Terminology.—The skull may be said to be fractured in distinction to its being wounded when, as the result of a blow, it becomes cracked or broken into more or less separate pieces. These injuries are classified in a variety of ways:

(*a*) According to the mechanism of their production.

(*b*) According to the presence or absence of a communicating wound.

(*c*) According to the form assumed by the fragments.

(*d*) According to their situation.

(*a*) Depending on the mechanical factors at work in their production, they are distinguished as (1) *bursting fractures* and (2) fractures due to local depression or indentation—so-called *bending fractures*. In the case of fractures the result of penetration by modern high-velocity projectiles a further mechanical element comes into play, producing *expansion fractures* through the explosive force of hydrodynamic action.

(*b*) Fractures are *open* or *compound* when they are exposed by a wound of the overlying soft parts; they are *simple* or *closed* when the soft tissues covering them remain intact.

(*c*) According to their form they are distinguished as *linear* or *fissured fractures* when the bone is merely cracked without displacement; as a fracture by *diastasis* when there is a simple separation of the sutures; as *comminuted* or *fragmented* fractures when the lines of fracture intersect, so as to isolate separate particles of bone; as depressed fractures when fragments of bone, whether of the entire cranial thickness or of the inner table alone, are driven below their spherical level; as *perforating fractures* or *fractures* with loss of substance when they are the result of punctured wounds or when the fragments at the seat of the penetration have been carried away, leaving a defect, as is the case in most penetrating bullet wounds. And of these chief varieties many subdivisions may be made.

(*d*) Lastly, depending roughly on their anatomical situation, they are distinguished as *fractures of the base* and *fractures of the vault*,

[1] (Part I from Dr. Harvey Cushing's contribution to *Surgery, Its Principles and Practice*. Edited by W. W. Keen, M. D.; published by W. B. Saunders Co.)

and although the two are often combined they may exist separately and have different characters. Thus, fractures of the base are usually linear and their fragments, if comminuted, are rarely displaced, for the base is much less accessible to direct injury; hence, fractures there are usually the indirect result of violence applied elsewhere. On the other hand, the vault is directly exposed to injury and local comminution with dislocation of fragments is common, and as the bone is thick and has two determinable layers there are special influences which modify the character of the fragmentation.

The mechanism of fractures—Bending, bursting, and expansile fractures.—Regarded as a hollow shell of bone which possesses elasticity sufficient to rebound when dropped, the cranium must needs differ from all other bones of the skeleton in the mechanism of its injuries. Certain of the physical laws which explain the peculiar form assumed by these injuries are known to us; others are still in dispute, and though, from a strictly clinical point of view, of chief importance is the knowledge that under certain conditions breaks occur in a certain manner and lead to certain complications, we naturally search for an explanation of the reason why they so occur, even though this information may in no wise affect our diagnosis, prognosis, or treatment.

Teevan, Wahl, Rauber, Félizet, Aran, Bohl, Bruns, Bergmann, Kocher, and a host of others have undertaken clinical and experimental investigations directed toward the elucidation of the underlying principles governing cranial fractures.

We must take into consideration the double effect of an impact, for the blow may produce (1) disturbances which are direct and chiefly of local consequence, and (2) those which are indirect and lead to solutions of continuity at a distance. Setting aside for the moment its irregularities and considering the skull to be an elastic globe, an impact will momentarily lessen its diameter in line of the blow, and force nearer together the point or pole of impact and the point on the sphere diametrically opposite. As the impact forces the poles together it will at the same time bulge out the sides of the sphere and thus increase the equatorial circumference and, in a lesser degree, the circumference of all the other circular planes which lie perpendicular to the polar diameter. If the distortion following the impact is inconsiderable, the skull, owing to the elastic rebound, will resume its former shape unimpaired. If the distortion, on the other hand, is so great as to overcome the molecular cohesion of the bony particles, they will be disrupted. This may take place (1) as a rupture or bursting of the bone in parts remote from the poles of impact where cranial dimensions have been increased to the point of overcoming tensile strength of the particles, and (2) as a local indentation at the

pole of impact where cranial dimensions have been diminished to the point of overcoming the local resistance of the particles to pressure. These two qualities of elasticity—tensile strength and resistance to pressure—have been the objects of special study by Rauber, who has shown that resistance to pressure is a third greater than tensile strength. This, however, does not mean that fractures are less likely to occur at the pole of impact than at a distance, for other factors come into play.

Local character of injuries through bending.—These fractures usually result from the sharp impact of a body with a comparatively small surface. Such a blow expends its force quickly and a rebound occurs before the form of the skull, as a whole, has been sufficiently altered to produce lesions at a distance. At the pole of impact the bone is broken and the displaced fragments do not resume their former position.

In spite of its greater thickness and vaulted construction fractures of this sort are more common on the exposed calvarium than at the inaccessible base. The character of the lesion, furthermore, is influenced by the structural peculiarity of the bone; namely, its two dense tables separated by a spongy diploë. Owing to this, an indentation which leads to a bending fracture will cause the inner table to splinter and give way before the outer. In consequence we not infrequently find fractures limited to the inner table—a circumstance known even to the earliest writers in medicine, who explained the phenomenon on the supposition that the inner table was more fragile or brittle than the outer one, hence the "vitreous" surface. Not until Teevan's studies was the process satisfactorily explained on the ground of tensile strength or cohesion of particles on the one hand and of resistance to pressure on the other. There is no simpler illustration than the oft-used one of a green stick broken across the knee. The cranial impact leads to a local indentation, which tends to pull apart the particles comprising the inner table and to drive together those of the outer. In certain rare cases the process may be reversed and the outer table alone suffer; this implies a blow from within. Both Teevan and Bergmann have given instances of such lesions; thus, after traversing the cranial cavity, a spent bullet may strike the inner surface of the skull and fracture the overlying outer table alone.

If the force of the blow has been expended by the time the inner table gives way, it alone suffers fracture; if it continues, the outer gives way as well, but in the latter case it is always to be borne in mind that the inner table splinters over a wider area than the outer. A lesion which is limited to the inner table alone can occur only in a skull well provided with diploë, and consequently in infancy and

old age the bone will usually give way throughout its entire thickness at the same moment. (Fig. 65.)

These bending fractures may be associated with little or no displacement of fragments; they may, on the other hand, lead to a marked depression whose floor is made up of firmly wedged fragments from the two tables. They, furthermore, are usually bounded by an irregular circular fissure, into which lines of fracture radiate from the central point of impact. An excellent example of such a circular fracture from bending occurs among the comparatively rare instances of this form of fracture at the base, when, as the result of a fall upon the buttock the impact is transmitted to the occipital bone through the spinal column, and the circular fracture more or less clearly surrounds the foramen magnum.

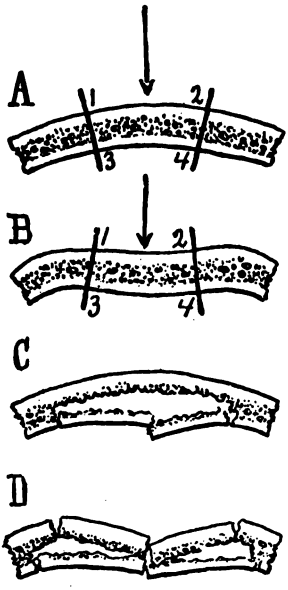

FIG. 65.—Illustrating mechanica principles of bending fracturesl A and B, arrows show direction. of impact [3 and 4 are dragged apart until tensile strength is overcome; 1 and 2 are crowded together until resistance to pressure is overcome (Teevan)]. C shows possible effect on inner table alone. D shows possible effect on both tables.

Distant effects of injuries through bursting.—A diffuse blow from a flat surface is prone to cause effects at a distance, just as a concentrated one from a small body is apt to produce local effects. A bursting fracture of typical form, comparable to the lesions, which Von Bruns has produced experimentally by compressing skulls in a vise to the point of fracture, was cited in the clinical note elsewhere, but it is unusual for the head to be caught and squeezed in this way. An analogous injury may occur when, lying on a hard surface, it is struck by a falling body, though a violent blow against one side of the cranium alone—the head itself usually being the moving force—is the more common method. Though the striking surface, favorable for a bursting effect, should be a flat one, it is common enough for some forms of impact, which produce primarily an indentation, to cause a bursting of the skull as well, in cases there is no immediate rebound and if the force exerted be sufficient. Thus, we often find meridional fissures which radiate from a local bending fracture situated at the pole of impact, or, indeed, even in the absence of a polar fracture. (Fig. 66.)

Thus most fissured fractures are an expression of the indirect or bursting effect of a blow, and inasmuch as the base of the skull is

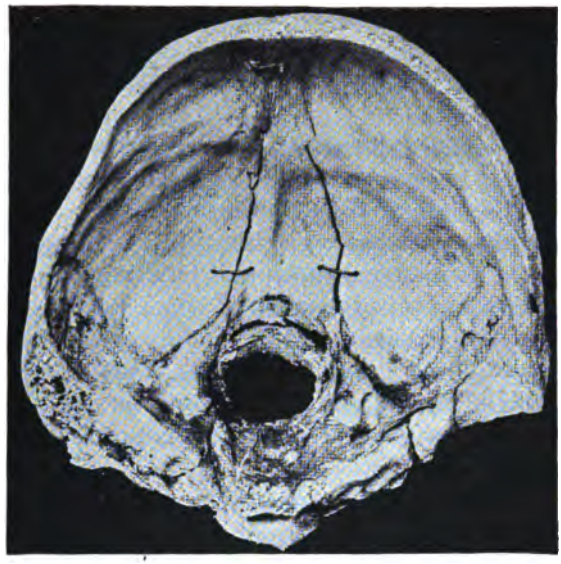

FIG 66.—Fissured fractures at the occipital base, from bursting, not involving foramen magnum. (Warren Museum.)

FIG. 67.—Base of skull showing typical bursting fracture passing through middle fossæ and sella turcica.

more fragile than the vault these fissures occur most readily in this region.

Having oftentimes no apparent connection with any lesion at the point of impact they naturally are spoken of as the indirect result of violence.

The view that these injuries at a distance are due to the effects of a counter-injury or *contre-coup*—a term introduced by French surgeons in the latter half of the eighteenth century—is one which remains popular, though it has been shown to be mechanically wrong.

We learn from these observations that bursting fractures need not be associated with any displacement of bone, but that linear cracks occur which have a tendency to run into the nearest weak portion of the cranial base. These cracks or fissures enter the middle cranial fossa more often than the anterior or posterior, and it is Walton's view that they often seek out the sella turcica, which presumably is the weakest point of all.

There are factors other than those already mentioned which are thought to modify the direction of these cracks from bursting. Among them are the foramina and the sutures. Whether the fractures tend to seek or to avoid the basal foramina is a matter of dispute. It seems to depend upon the relative strength of the rim and of the foramen and the neighboring bone. When the rim is thickened and strong, even so large a defect as the foramen magnum need not be an evidence of local weakness; no more need a trephine opening in the vault in any way weaken the elastic strength of the cranial sphere. Nevertheless, there are certain foramina which are apt to be involved, as the posterior lacerated space, the foramen ovale, and the facial and acoustic foramina.

The sutures, on the other hand, often serve to deflect fissures from the direction which they should have taken by mechanical laws. This is especially true for the skulls of young individuals. (See Fractures by Diastasis, p. 126.)

The explosive effect of hydrodynamic force.—The introduction of the modern firearm, with its peculiar nondeforming, hand-mantled projectile, has brought an entirely new element into the mechanism of penetrating gunshot wounds, particularly those involving the cranium. It has been made the subject of special study by Kocher, von Bruns, Coler, and Schjerning. When such a projectile, with its extreme initial velocity and great penetrating power, traverses the incompressible semifluid brain, inclosed as it is within a solid covering, it exerts an enormous explosive (hydrodynamic) force against the inner cranial surface. Were the cranial chamber empty a simple penetrating wound of entrance and exit would result, but, being full, the tremendous force is transmitted against all points of its inner surface, and consequently its walls become shattered into fragments.

(*b*) **Clinical varieties of fracture.**—By the qualification *simple* or *compound* or, possibly better, *open* or *closed*, we indicate as in skeletal lesions elsewhere, that the fracture is covered by intact soft parts or communicates with the air through an external wound. The distinction is possibly of less vital significance than formerly and, indeed, here more often than in any other part of the body we deliberately, by operative explorations, turn simple fractures into open ones for the replacement of dislocated fragments or to avoid other complications. In compound fractures of the base, however, we are almost as helpless in the prevention of infection as were our predecessors; for when these injuries communicate with the ear, the pharynx or the sinuses accessory to the nasal cavity, where pathogenic organisms lurk, a doorway which we can not reach is opened to infection. A compound fracture of the base from other cause than bursting is unusual, though it may occur when a weapon, a bullet, or other missile has entered the skull from below. In such a case careful surgical cleansing and drainage is demanded.

Fractures according to their form.—Fissured or linear fractures, as we have seen, are the usual result of bursting; they tend to take a meridional course, radiating from the pole of impact, and, further, owing to its structural weakness, they more commonly occur at the base. When the skull resumes its former shape, after the moment of deformation which causes the bones to spring apart, the fissure will close tightly, provided there has been no associated fragmentation. At the moment of separation of a fissure, substances like hair, portions of headgear, or pieces of the missile which inflicted the blow may either be introduced wholly into the cranial chamber or be caught in a vicelike grip when the edges again snap together. It acts like the meridional crack in a child's hollow rubber ball, which gapes when its poles are compressed.

A fissured fracture may occur as a single linear crack, it may fork or branch, or there may be mutiple fissures radiating from the point of impact. A simple linear fissure may close so snugly as to be difficult of detection even on direct exposure. Attention may be called to it, however, by the extrusion, along the closely approximated edges, of fine drops of blood. After death this does not help and at autopsy fissures may escape other than the closest scrutiny. In other instances, whether from interposition of tissue or from some dislocation of fragments, an extensive meridional crack may continue to gape. Such a condition, especially when the fissure has included the vault, may be detected by percussion, or when the head has been shaved, by auscultation combined with percussion, the blow eliciting a "hollow-cask" sound. Furthermore, there will be tenderness along the line of fracture, though this is of little aid in unconscious pa-

tients. It is to be remembered that sutures are often mistaken for fissures.

Linear fractures, though simple in themselves, are especially prone to be accompanied by intracranial complications, for their very presence indicates a diffuse blow the effect of which is usually widespread. Hence cerebral contusions are common. The fissures often run across the meningeal grooves and lead to extradural extravasations and more or less subdural hemorrhage is the rule in the linear fracture of the base.

The treatment, therefore, resolves itself into the treatment of the complications rather than of the fracture, viz., the evacuation of the clot in an extradural hemorrhage; the drainage of the cerebrospinal space if subdural hemorrhage or edema has been sufficient to cause bulbar symptoms.

Fractures by diastasis.—Linear fractures may be deflected into one of the sutures, due to the fact that, before the complete obliteration which they may undergo late in life, they offer less resistance to the cranial deformation than does the bone elsewhere. In the young the lesion may occur as a true separation of the bones. I have seen at operation upon a child of 12, whose head some days before had been caught and laterally squeezed, a simple diastasis of the coronal suture, which had torn the dura, leading to the escape of cerebrospinal fluid under the scalp (spurious meningocele); at no time had there been any cerebral symptoms. In the adult, on the other hand, the process is necessarily more than a simple diastasis, for union is so firm, owing to the close dovetailing of the irregular bony margins, that separation of the sutures can not occur otherwise than by a break.

Comminuted fractures are those characterized by more or less fragmentation or splintering. The comminution may be confined to the area of impact or the entire cranium may be broken into pieces. Being a common result of local deformation or inbending at the point of injury, they are usually situated on the vault and depend for their production on the character not only of the blow (a sharp one with quick rebound) but also of the striking body.

From the standpoint of the bony lesion itself they are more serious than linear fractures, owing to the usual displacement of fragments—*fractures with depression*. Extensive comminution, however, may occur with little, if any, dislocation of the broken pieces. We may, furthermore, in the elastic skulls of infants have depression with no comminution or, indeed, with a total absence of fracture. Finally, in certain rare cases, fracture may occur with actual elevation of a fragment. These effects, however, are unusual; comminution and

depression commonly go hand in hand. Hence they will be considered together.

The comminution and depression may affect the inner table alone or both tables, in which case the fragments may consist of the entire thickness of the skull or, in diploëtic skulls, of the separated tables. When thus separated the fragmentation of the inner is always more widespread than that of the outer. The fragments may form a cup-shaped depression, often termed by English writers "pond fracture," or they may become tilted at the periphery and slip under the intact cranial edge. We thus have *peripheral* or *central depressions.*

From the pole of impact in comminuted fractures there are often numerous radiating or meridional fissures; these in turn are often connected by zonal lines of fracture, like the connecting strands of a spider's web; and in these cases the farther from the point of impact the farther apart are the zonal lines, and consequently the larger the fragments. When comminution is the result of diffuse blows, as in the skull of the "butting" negro in the Surgeon General's Museum, or when it follows falls from a great height, irregular fragmentation, like a broken eggshell, may occur, with fissures having no definite configuration.

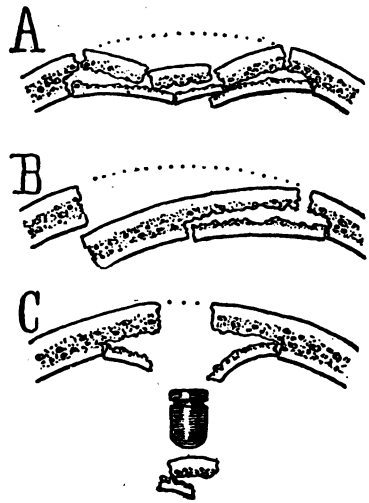

FIG. 68.—Diagram of (A) comminuted fracture with central depression; (B) one with peripheral depression; (C) one with loss of substance.

Almost all punctured or penetrating wounds are accompanied by more or less local fragmentation, with depressions, which particularly affects the inner table.

Depressed fragments may heal in place and their irregularities become, in the course of time, largely smoothed off.

Perforating fractures are due to cuts, to stab wounds, to the penetration of sharp tools which have fallen from a height, to the blow of a pick, the thrust of a bayonet, and what not. They are associated with more or less fissuring, with fragmentation, and with depression of fragments, especially of those broken from the inner table about the margin of the wound. Their course, diagnosis, complications, and treatment do not differ materially from that of wounds of the skull, unassociated with fracturing, though produced by similar agencies. When a portion of bone has been carried away, leaving a defect, they are called *fractures with loss of substance.*

One particular group of perforating fractures deserves special consideration, namely, those which are the result of wounds from firearms.

Gunshot fractures.—In their simplest forms these are perforating fractures which produce a circular loss of substance. When the result of a wound at short range from the modern small arm, we have seen that the skull may be burst outward by the explosive action of hydrodynamic pressure. We have learned, too, that these are compound or open fractures; that they are almost always comminuted

FIG. 69.—Showing irregular fissuring (also diastasis) from explosive effect of through-and-through gunshot wound. Note meridional cracks radiating from pole of impact. (Surgeon General's Museum.)

ones with some depression of fragments; and, finally, that they often lead to sepsis, hemorrhage, or other intracranial lesions, which make of them a particularly dangerous and crippling form of injury.

There are, however, other types of gunshot fractures less serious, since they are nonperforating. Thus, the direct impact of a heavy spent ball may fissure or indent the skull without producing more than a bruise of the scalp. Again, a bullet may pass through the scalp and graze the vault in a tangential direction without penetration, or it may furrow the bone, scooping out a gutter in the outer table alone or leaving a defect of the entire cranial thickness. The

lateral force exerted, during its rapid flight, by the modern high-velocity projectile is sufficient to comminute the skull, even if it be merely grazed, so that only in those localities where the bone is thick and porous or contain air cells would it be likely to escape considerable local fragmentation from such a tangential wound. For the same reason fractures of the base may occur when a bullet traverses the shell-like bones comprising the undersurface of the skull without actually penetrating the cranial chamber; and, further, bullets may become lodged in the thicker parts of the cranial wall and produce more or less local comminution without actually entering the cavity.

The wound of the bone, as we have seen, may show nothing more than a clean-cut circular or oval loss of substance, but it is the rule for the wounds both of entrance and exit, in case they occur in bones containing diploë, to show the particular characteristics of punctured fractures; that is, to have a more or less splintered margin, particularly of the table which has been last penetrated—the inner table for the wound of entrance, the outer table for that of exit. Hence, when there is a loss of substance due to the actual carrying away of fragments, the circumference of the defect will be greater on the side from which the missile has emerged, whether it be wound of entrance or exit—a matter often of medicolegal importance. Furthermore, meridional fissures are apt to radiate from the wounds of impact, and these meridians in turn are often joined by circular fissures on zonal planes.

The damage from perforating bullets depends partly upon the physical properties of the missile and partly upon the speed with which it is traveling. There is great difference, therefore, between the effects of the soft, leaden bullet discharged from a revolver and that of the modern conical projectile with its hard mantle and tremendous initial velocity. The latter missiles, except near the end of their flight, rarely lodge; the former almost invariably do.

In his *Handbuch der Praktischen Chirurgie*, Bergmann gave in full the results of experiments conducted by himself and others upon wounds of the head made by the modern rifle. Briefly, it may be said that at close range the skull and scalp are literally torn to pieces and the brain disorganized; that on penetration at 50 yards the scalp remains intact, though the skull is greatly comminuted and brain tissue oozes from the wound of entrance and of exit; at 100 yards there occur zonal fractures which tend to be limited to the area about the wounds of entrance and exit, while meridional fissures radiate from these points, showing that explosive action is still effective; at 1,000 yards the zonal cracks encircling the bullet holes disappear, and only the radial fissures remain; at the distance of 1 mile the fissures largely disappear, leaving the two clean-cut bullet

holes; and not until over 1½ miles does the projectile fail to emerge after entering the skull on one side. All this, of course, is merely relative, for there would be great difference, not only in individual skulls, but in the position in which they were struck; and it is, after all, a matter chiefly of interest to the military surgeon.

The gunshot fractures which are seen in civil life are more apt to result from revolver shots and in orderly communities to be self-inflicted wounds, whether by accident or intent. Here again the character of the injury depends upon the nature of the weapon and the initial velocity of the projectile. Most of the wounds which one sees today are produced by soft bullets fired from the ordinary revolver with no great initial momentum; the heavier army pistols, on the other hand, fire a projectile which at near range has the explosive effect of a rifle. The soft, deforming revolver bullets are apt to lodge either in the bone at the site of entrance or somewhere within the cranial cavity, either at some point in the direct line of their flight or, in case the missile has rebounded, at some point in a line determined by the angle of deflection from the opposite inner surface. At times the course of such a deflected bullet may be mathematically calculated, but today the X-rays give us a more accurate means for determining its position. It may be said, however, that, as a rule, there is no particular reason for its extraction, for in the absence of immediate complications it becomes encapsulated and, unless the missile chance to lie near the surface, the damage already done will only be increased by meddlesome attempts to locate and extract it.

The complications which result from penetrating bullet wounds may be classified as *immediate*, or those due to hemorrhage, compression, and destruction of tracts; *intermediate*, or those due to sepsis; and *late symptoms* (irritative and paralytic), giving evidence of the permanent damage done to the cerebral tissues. Hemorrhage, of all the immediate symtoms, is especially to be dreaded, as it may lead to rapid death from compression. In all cases in which there is an increase in intracranial tension from effused blood, the pressure forces the disorganized nervous tissue through the wounds of entrance and exit, and the extruded particles of white tissue are found mingled with the blood which oozes from the opening. It is not uncommon for cranial nerves to be injured, either by direct section or by implication in a basal fracture.

It is notorious that suicides often fail to accomplish their purpose. Brun has recorded 32 cases, of which number 16 recovered. Of these cases the wound of entrance was in the right temporal region 16 times, in the left twice, in the forehead 9 times, and in the mouth twice. The "temple" is regarded by the laity as a particularly vulnerable spot, which accounts for the preponderence of

the attempts in this situation. In them oftentimes the bullet merely passes extra-cranially from temporal fossa to temporal fossa, through the back of both orbits, cutting the optic nerves and leading to blindness—a sad penalty for a criminal act. Though unrecorded in Brun's series, suicidal wounds inflicted in the right mastoid region are not uncommon.

The later complications, in case of "recovery," are paralysis, mental changes, epilepsy, etc. Thus, a patient was accidentally shot in the mid-line of the forehead at the hair margin. A surgeon removed two pieces of the bullet (supposedly all of it), together with some fragments of bone at the wound of entrance, which finally healed. The patient for a time was aphasic, had a left-sided hemiplegia, and he subsequently developed epilepsy with a peculiar speech aura. An X-ray plate then showed a foreign body lodged in the left side of the brain near the skull, and just below the middle of the Sylvian fissure. An operation was performed, adhesions due to an absorbed subdural clot were separated, and a small, dense scar, inclosing the main fragment of the bullet, was removed. This had traversed the left frontal lobe, had struck the side of the skull, and ricocheted into its position. The extraction of the bullet benefited him in no respect, and he is progressing to mental degeneracy.

Fractures according to their situation—Fractures of the vault and fractures of the base.—There is a certain justification in this common anatomical division, for in the two situations not only do fractures differ in the mechanism of their production but also in the form which they assume and in the complications to which they are liable. The bones of the exposed vault are more liable to direct, indenting injuries, and hence, despite their greater strength, comminution with dislocation of fragments is frequent; those of the well-protected, though more fragile base, are more subject to fissuring, the result of general deformation of the skull. These, of course, are not invariable rules, for we may have simple fissures of the vault from bursting or a local comminution with depression at the base from bending—an example of which is the not uncommon fracture which occurs about the foramen magnum as the result of falls on the buttock when a direct blow is transmitted to the base through the spinal column. Again, a simple bending fracture of the base may follow a sharp blow on the chin, when the ramus and condyle of the jaw transmit the force to the base—the prize-fighter's fracture. Finally, it is always to be kept in mind that fractures of vault and of base are apt to be associated.

Many statistical studies in regard to cranial fractures have been made from time to time; notable among them is the recent elaborate monograph of Hans Brun, based on 470 cases which in 20 years had been carefully observed in the Cantonal Hospital in Zurich.

Occurrence.—In general it may be said that they are injuries of young adult life; that they are many times more frequent in men than in women; that in the majority of cases (60 per cent) they are the result of falls from a height. About one-half of these fractures involve the base, often alone, sometimes with associated fracturing of the vault. On the other hand, it is estimated that 70 to 75 per cent of all fractures of the vault are accompanied by basal injuries. Fractures of the vault are more often compound than simple, and they occur with about equal frequency in frontal, parietal, and temporal bones, being rare in the occipital region. Basal fractures are more common in the midcranial fossæ.

Mortality.—Disregarding the etiological factor, the patient's age, and also the character of the injury, about one-third of all cases in the past have proved fatal, and as the fatalities are largely due to the immediate cerebral complications, modern methods of treatment have not served to greatly alter these figures. The percentage of fatalities increases with age—the younger the individual the more favorable the outcome. Fractures of the base are commonly thought to be attended with a higher mortality than those of the vault, though with our improved diagnostic measures (lumbar puncture, for example) we may find that many cases of simple basal fracture have heretofore been overlooked and regarded merely as concussion—a fact which may make one's percentage of recoveries at least appear larger to-day. Excluding those cases which have died as an immediate result of the injury and those which have later succumbed to infection, the average duration of life in the fatal cases is said to be 44 hours; so that there is some basis for the old rule, adhered to by Bergmann and Wagner, that survival over two days gives a favorable prognosis.

Of the cases which survive the first 48 hours, a considerable number (8 per cent of all fatalities) die from the intermediate complications of meningitis or abscess. Fractures of the base are more liable to this complication than those of the vault; for the latter are accessible and easily drained, so that, unless there be a defect leading to a fungus cerebri, dangerous from a persisting leak of cerebrospinal fluid, infection rarely occurs. Basal fractures, on the other hand, especially those which open up the sphenoidal or ethmoidal sinuses where pathogenic organisms lurk, are often followed by a meningeal infection. In this case the pneumococcus is the more common agent, whereas in fractures of the vault a streptococcal or staphylococcal infection is the usual one. I have twice seen a rapidly fatal pneumococcal meningitis start up the third day after what appeared to be a simple, uncomplicated basal fracture with a little bleeding from the nose and so few subjective symptoms that the patients remonstrated at their enforced recumbency.

The *prognosis* is in no way proportionate to the extent of the cranial injury, but depends entirely on the character of the intracranial lesions which will be fully considered anon. An insignificant crack of the base, associated with a focal hemorrhage in pons or medulla, may put a sudden end to life; whereas an extensive fragmentation of the vault, which allows for considerable cerebral expansion, may actually save life through "decompression." An insignificant punctured fracture which does not even produce concussion may prove fatal from meningitis or abscess later on; a comminuted and depressed compound fracture may, on the one hand, cause death quickly from hemorrhage and compression, or may heal practically untreated and give few symptoms. On the whole, the immediate prognosis is more favorable in bending fractures than in bursting fractures; in other words, more favorable in those which are accessible than in those which are not, for it depends largely on the possibility of early treatment not only of the fracture, but of its underlying complications.

The *diagnosis* of fractures of the vault may offer difficulties, particularly in the case of linear fissures and of those involving the inner table alone. One, however, is much more apt to be misled by the peculiar feel of the infiltrated edge of a subaponeurotic extravasation into making a faulty diagnosis than to overlook a cranial fracture when it is actually present. When the scalp is intact, linear fractures may at times be recognized through lines of tenderness on pressure, particularly over the temporal fossa, and by a changed percussion note if there is any gaping of the fissure. In open wounds there should be no difficulty in recognizing even a closely approximated fissure, owing to the blood which oozes from between its edges; sutures, however, may be mistaken for fissures. Localizing cerebral symptoms, to be discussed later, are often helpful in determining the situation of a depression if it is not actually palpable, and involvement of cerebral nerves may indicate the direction taken by a meridional fissure. Old deformities dating from birth, patches of senile atrophy, defects from former diseases, like syphilitic osteomyelitis, and the irregularities present in all skulls, though more marked in some, may at times be mistaken for depression.

In fractures involving the base alone we must, in the long run, depend entirely upon the symptoms which we have learned to recognize as common accompaniments of these injuries rather than upon any direct evidence of the bony lesion. Evidence from intracranial or extracranial bleeding, either free or into the tissues, is of particular value.

The intracranial extravasations usually take place into the subdural space, for, owing to its close attachment, the dura is usually

torn when the bones are fissured. The amount may be small or so extensive as to cause rapidly fatal compression. It may be recognized by finding evenly distributed red blood corpuscles in the cerebrospinal fluid withdrawn from the lumbar meninges.

The extracranial extravasations may also be free and bleeding may occur from the nose, mouth, or ears, in case the ethmoid, the accessory sinuses, the Eustachian tube, or the tympanic cavity have been implicated. It is necessary to exclude a simple "bloody nose," rupture of the tympanum, or entry of blood from without into the auditory canal. Extravasations into the tissues (ecchymoses) appear more tardily. They are common in the orbit, under the eyelids or conjunctiva when the frontal plate is injured, and in fractures of the middle or posterior fossæ they find their way to the surface over the mastoid process or down the neck after some days.

The escape of cerebrospinal fluid often occurs with fractures, entering the middle fossa, particularly when they involve the petrous bone and when both dura and tympanum have been torn. The escape of bloody fluid may continue for days and the sympton need not always be entirely undesirable, as pressure may be relieved thereby. Open fractures which communicate with the nasal or pharyngeal cavities may likewise be followed by a leakage of cerebrospinal fluid, though it is less common from these situations. In rare cases, after a lesion of the petrous bone unaccompanied by rupture of the tympanic membrane, fluid may escape into the pharynx by way of the Eustachian tube and either be swallowed or flow from one nostril when the head is tilted down.

The *complications* of cranial fractures often serve as an aid in diagnosis. They are estimated to occur in 46 per cent of fractures of the vault and in 64 per cent of those of the base. Varying grades of concussion, contusion, or compression are almost inevitable; only exceptional forms of fracture occur without one or another of these classical symptoms, though any one of them may result from an injury in the absence of fracture. They are apt to be more outspoken in basal lesions, owing to the diffuse character of the blow necessary to produce a bursting fracture. As will be described in its proper section, compression may be general or local, and when local it may give cerebral symptoms of irritation or of paralysis, which serve to point out the situation and character of the cranial lesion. This is often the case with indented fractures of the vault which lead to cortical laceration of the brain, or with meridional fissures which cross the meningeal groove and lead to extradural hemorrhages with their characteristic "interval" between symptoms. These extradural hemorrhages can occur only under the vault where the dura is more easily separable from the bone than from the base; and it is to be remembered that they are not necessarily an indication of fracture, but may

be the result of simple deformation not sufficient to break an elastic skull.

Involvement of the *cranial nerves* may prove a valuable diagnostic aid. The facial is by far the most commonly injured, owing to its devious course through the petrous process, so frequently implicated in fissures entering the middle fossa. The mere presence of hemifacial palsy, however, after an injury to the head need not indicate with certainty the peripheral involvement of the nerve, for it may be due to a contralateral central lesion. In order of frequency the abducens comes after the facial, and diplopia from an involvement of any or all of the oculomotor nerves may be the result of breaks in the neighborhood of the sphenoidal fissure. In fissures crossing the frontal fossæ the olfactory often suffers. The optic may be affected by direct injury and lesions of the trigeminus, glossopharyngeal, vagus, spinal accessory, and hypoglossal have been recorded. The nerves are apt to be affected in groups, the seventh, eighth, and sixth together; the fifth and third; or the ninth, tenth, and eleventh. The lesions usually occur at or near their foramina of exit, due either to actual laceration or to local compression from effused blood; and hence a study of the paralyses may indicate the fossa which the fracture has entered, whether anterior, middle, or posterior.

Serious complications from hemorrhage may follow *injuries to blood vessels*, particularly when the sinuses or meningeal artery are lacerated, and occasionally linear fractures crossing the middle fossa toward the pituitary fossa may so traumatize the cavernous sinus and carotid as to produce an arteriovenous aneurysm with pulsating exophthalmos.

The sequels heretofore considered are common to all lesions of the skull; those due to an *infection* are almost without exception limited to open fractures, whether of base or vault. In them purulent cellulitis, osteomyelitis, septic sinus thrombosis, meningitis, or cerebral abscess were formerly almost to be expected. Modern methods have largely lessened these evils in the case of the vault, even if not in basal lesions.

An insignificant fissure which passes across the ethmoid plate may open a pathway of infection from the nasal cavity and lead to a rapidly fatal meningitis. Cerebral abscess is especially common after punctured or gunshot wounds from the deep inoculation of infective agents, though it occurs often enough in compound comminuted fractures which have led merely to a superficial laceration of the cortex.

Rarer complications, like spurious meningocele, pneumatocele, and others too numerous to mention, may likewise occur. Cysts occasionally form after fractures either from a torn dura, from the partial absorption of a clot in the subdural space, or from a subcortical

extravasation. Sugar may appear in the urine (traumatic glycosuria), usually about 8 to 12 hours after the injury and in about 9 per cent of all cases, according to Higgins and Ogden. All of these, as well as the so-called post-traumatic neuroses, result from the cerebral—not from the cranial—injury.

The *process of healing* does not take place as in the long bones, where there is an abundant callus formation. Dura and periosteum, however, are both capable of forming new bone, as we have seen in exostoses, osteophytes, etc., and complete repair, even when there has been a loss of substance, may occur. It may, however, be long delayed or may completely fail, due, according to Bergmann, to the destruction or loss of the osteoplastic layer of both inner and outer periosteum as well as to the absence of movement which ordinarily stimulates callus formation.

Union often occurs by fibrous membrane alone; even narrow fissures may fail to become reunited by bone. As a rule, however, a slow process of bone production and bone absorption goes on, hand in hand, and irregular edges or depressed fragments are rounded off as the gaps are more or less filled in. Occasionally there is an overproduction of new bone, leading to focal or to widespread hyperostoses from either the outer or inner table. Even defects of some size may at times become entirely ossified, and even when closed by membrane alone they may become so firm and inelastic as to show no pulsation. According to Bergmann, defects can not be expected to close if they exceed a diameter of six to eight centimeters, and it would indeed seem that it is rare even for much smaller openings to fill in.

There is a great difference of opinion as to the injurious effect of these bone defects, some holding the view that when extensive they lead, in the course of time, to serious mental symptoms. Cushing does not believe that they are injurious unless accompanied by an underlying lesion of the dura. When the dura is wounded and the scar formation leads to adhesions between overlying scalp and brain, the chronic fibrous changes which result may lead in time to extensive cortical alterations and mental deterioration. With an intact dura, however, such symptoms are less likely to occur.

Treatment.—We are confronted again by the necessity of distinguishing between the management of the fracture itself and the management of its complications. Relatively simple rules can be laid down for the former; for the latter our conduct is largely controlled by physiological laws relating to the circulation of the blood and cerebrospinal fluid under abnormal conditions. In fractures of the vault the indication for surgical intervention is usually deformation of fragments, rather than critical cerebral complications; in

fractures of the base it is the reverse, for there intracranial complications are especially serious and deformation is rare.

In compound injuries of the vault we may easily determine the form and estimate the consequences of the injury, and our endeavor should be to thoroughly cleanse the wound, to elevate depressed fragments, to restore a wound in the dura if one exists, and to leave the parts as nearly in their mutual position as possible. If the fragments are depressed and wedged it may be necessary to trephine at the edge of the depression before they can be pried into place. Even in the absence of visible depression an opening may be required when cerebral symptoms are present, due to depression from the inner table alone or to intracranial hemorrhage.

It is another matter when injuries of the vault are covered by intact scalp, for there may often be great difficulty in determining whether there is sufficient justification to transform a simple into a compound fracture, even for the sake of determining the lesion. It is largely a personal matter and rests with the judgment of the operator; and this in turn depends entirely upon his familiarity with intracranial disturbances which are amenable to operative treatment and his ability to safely cope with them when found. A simple fissure which crosses the temporal region of itself needs no surgical intervention, but this is urgently called for when pressure symptoms indicate either a lesion of the meningeal or free extravasation at the base. When simple fractures are accompanied by evident depression surgical measures are indicated, even in the absence of immediate cerebral symptoms, for unelevated fragments are almost certain to be the source of future trouble, especially if the dura has been injured. The opening must be carefully examined on all sides, for depressed fragments are readily overlooked. In Fig. 50 (Keen's Surgery, Vol. III) is shown a patient with a depressed fracture which had solidly healed, in so far as the union of the displaced fragments was concerned, and the rounding off of their sharp edges, and yet serious mental (left frontal lobe) symptoms resulted. In an old healed depression of this sort it is necessary to remove the entire area by a circular incision and either to leave a defect, to replace it by some foreign material, or to cover it by an osteoplastic flap. Occasionally the cup-shaped area may be replaced inverted without subsequent necrosis.

When there is extensive comminution with many loose fragments it may be difficult to determine whether any of them should be removed, owing to possible loss of viability. The dread of necrosis and of infection, though a natural one, is largely an inheritance from our surgical forefathers, and it is a matter of present-day experience to find that fragments of surprising size, even when completely separated, will survive if left in a clean wound. It formerly was the

custom to remove all completely detached pieces. It has been learned, however, that even boiled fragments may heal after reinsertion. Cushing has quite frequently replaced a 3-cm. trephine button after boiling it (owing to some suspicion of its perfect cleanliness), and has never seen it fail to heal in place; it is well known, of course, that an unboiled fragment of this size can always be safely replaced. In the latter case it is probable that bone-forming cells of either surface or of the diploë may remain viable, but in the former instance they have been destroyed and it is to be presumed that the fragment acts only as a temporary stimulus for new tissue, becoming itself ultimately absorbed. The question of closure of defects will be considered later.

The treatment of basal fractures resolves itself largely into the treatment of contusion or compression of varying degrees, for which therapy is largely restricted to rest, absolute quiet, an ice cap, sedatives when headache is severe or when there is great restlessness, and to free evacuation of the bowels, preferably with a saline—measures to be observed in practically all cases of cranial injury. The greatest care should always be exercised in handling and in transporting any case of fracture with intracranial symptoms, for the symptoms are much aggravated by any form of jolting.

With the view of preventing infection in case there is bleeding or loss of cerebrospinal fluid from the nose or ears, it is customary to irrigate and tamponade the auditory meatus or the nasal chamber. This procedure, however, can be overdone, and only in case the hemorrhage is profuse is it justifiable to actually pack these orifices, for nothing is more certain to set up a suppurative infection of the mucous membrane. Irrigation of the nose or of the ear to remove clotted blood and to cleanse the cavities has an element of danger, and it is preferable to accomplish this by merely wiping out the passages with a sterile cotton swab moistened in a mild antiseptic solution. It is important not to irrigate the Schneiderian membrane in such a way as to produce a sneeze, for on more than one occasion this has been disastrous and the explosive effect has driven septic material into the middle ear or ethmoidal cells and apparently has been the active agent in inaugurating a septic meningitis. After cleansing, the cavities should be loosely closed by a wisp of absorbent cotton, or of iodoform gauze if desired, which will serve to take up the secretions and which should be frequently changed if there is abundant discharge. Although by energetic measures we may overcome a local meningeal infection which has started over the hemisphere, we stand practically helpless before one originating at the base, although suboccipital drainage in a few cases has apparently resulted in cure.

One can speak somewhat more encouragingly in regard to active interference in case of diffuse hemorrhage. The fatalities from this cause, as will be detailed in the section dealing with compression, are due to a final implication of the vital centers in the medulla when the amount of effused blood is sufficient to so increase intracranial tension that they are thrown out of function from anemia. Though this has been well recognized, operative methods of meeting the situation have been inefficient or untried, owing to the feeling of hopelessness in the presence of continual oozing from an inaccessible and often uncertain lesion. Exploratory openings have usually been made over the vault, but, owing to the increased cerebral tension, such openings become filled with a bulging brain, drainage can not be effective, and a fungus cerebri is often produced.

It is self-evident that an opening as near the lesion as possible is desirable, and, inasmuch as most of these fractures enter the middle fossa, an opening low down in the temporal region is most likely to be efficacious. The author's procedure, which has been designated as an intermusculotemporal operation, often meets the needs of the condition. In this operation, by splitting the temporal muscle in line of its fibers and by rongeuring away the thin squamous wing of temporal and adjoining sphenoid, not only is the region overlying the meningeal vessels exposed, so that a chance extradural hemorrhage can be brought to light, but also the dura over the temporal lobes is exposed and the presence or absence of subdural effusion can be determined. The dura should be opened and there will usually be an abundant escape of bloody cerebrospinal fluid, whose evacuation will be aided by passing a curved, blunt dissector down under the temporal lobe. Oftentimes edema, as we have seen, has played the chief rôle in the compression, and we may find that merely a so-called "serous meningitis" is present, and that evacuation of a large amount of fluid will diminish the tension. In such case the muscle and scalp may be closed, but if there is continuous bleeding it is well to close the muscle only in part and to leave, at its lower angle, a strip of rubber protective, leading under the temporal lobe as a drain. In case the craniectomy on one side alone seems insufficient a bilateral operation may be performed at the same time or at a subsequent sitting, for the procedure is simple and not attended by shock. Its advantages are due to (1) the frequency of the bony lesion in the middle fossa; (2) the fact that cerebral contusions are especially liable to involve the tip of the temporal lobe; (3) the exposure of the meningeal territory and ease of determining the presence of an extradural hemorrhage; (4) the possibility of draining through a split muscle rather than directly through the scalp; and (5) the subsequent protective action of the muscle in

case a hernia tends to form in consequence of traumatic edema. The unilateral or bilateral defect in this situation leads to no complications and no subsequent deformity.

The Röntgenographic examination in fractures of the skull.—Anatomists describe the skull as "a superior expansion of the vertebral column as if composed of four vertebræ, the elementary parts of which are specially modified in form and size and almost immovably connected, for the reception of the brain and special organs of senses." The structure and dome shape with its double-deck of compact bone and intervening cancellated material, its ribs, bridging, and tressling, its thickened protuberances at prominent and susceptible points, the peculiar dovetailing of the sutures, the elasticity of the outer table, the overlapping of some bones and the density and mobility of the scalp, all combine to make this casing for the vital structures resilient to external violence.

Even with this reinforced construction, there are certain weak points which are particularly susceptible to fracture; namely, the vertical and orbital plates of the frontal bone, the middle cerebral fossa, the center of the squamous portion of the temporal bone, and the center of the inferior occipital fossa. A fracture, therefore, is generally directed toward the above points, although if the violence be sufficient, the ridges surrounding these areas may also be involved.

Because of its shape, elasticity, close fitting covering of skin, muscle, fascia and periosteum on the outside, and the pressure of the intracranial contents and the dura on the inside, fractures of the skull have a strong tendency to immediately replace and maintain themselves in position. For this reason, we find fractures indicating only slight bony injury, the grave lesion being to the blood vessels, intracranial nerves, dura and brain substance and the secondary conditions arising therefrom; the presence or absence of a fracture, in such cases, fades into insignificance except as a clue to the site of the injury and a guide for surgical procedure.

Varying from this class, we have the other extreme; namely, those having an extensive bony lesion, as in depressed or perforating fractures, with or without injury to the underlying structures; in such cases the location and character of the fracture must be considered. Here the force is consumed in breaking the bone at the point of contact, the effect being dependent upon the amount of force, the point of injury, and the character of the body producing the fracture.

Between the two definite forms above mentioned, there is much variance in the importance of the bony injury.

Fractures of the skull may be divided into those of the vault and those involving the base. In over 50 per cent of the cases examined röntgenographically, we find a combination of both. Fracture of the vault may be due to direct or indirect violence, or to an extension

from the base. We are familiar with Aran's theory of irradiation, the bursting theory, fractures by *contre-coup* and the Rawling theory of direct violence. It would seem as though each applied, as a causative factor, to certain classes of fracture of the skull. In our experience, those cases which are able to reach the Röntgenologist present a fairly constant effect of direct violence, the point of impact receiving the maximum amount of bone injury; from this point, we have radiating fracture lines which seek the weaker parts of the skull in the immediate neighborhood—this may be the vault, the base or both.

The common fractures of the skull are described as fissured, comminuted, diastasis of the sutures, depressed and perforated. The frequency with which these fractures are mistaken for various other conditions, especially in cases of coma, calls for any method which will make a rapid and positive diagnosis. This, in addition to many other difficulties which are encountered in the correct interpretation of this lesion, renders, in all head injuries, the routine Röntgen examination of the skull of the greatest importance. It offers an actual visual demonstration of the presence or absence of fracture, its location, character and extent.

Every skull receiving violence, imparted either directly or indirectly, with or without symptoms of intracranial injury, should be considered possibly fractured until proven otherwise. Such cases should receive a systematic Röntgenographic examination. If this procedure is carried out routinely, the relative frequency of fracture of the skull, as reported at the present time, would show a much larger percentage. We base this statement on the number of cases in which the diagnosis can be made only by means of the Röntgen examination, especially when a patient, with a history of a fall, has no symptoms, either objective or subjective. Experience has taught us that about 20 per cent of head injuries show fractures; this percentage was ascertained by examining a series of 300 cases, 60 of which were positive.

When a case of head injury is referred to the Röntgenologist for examination, the patient is often in an irritable, semi-conscious or comatose condition. Extreme patience and perseverance are required, and even these will not always succeed, making it frequently necessary to administer a hypodermic of Magendie's solution. It must be borne in mind that the *minimum amount of disturbance and movement is the rule.*

In skull examinations the patient's head must be absolutely fixed and all respiratory movement overcome. If the objective symptoms, such as bleeding from the ear, nose or mouth, laceration of the scalp, hematoma or paralysis be present, they are clues to the possible

site of the fracture and attention is naturally directed toward that area; this must not mislead one, however, for every examination should cover the frontal, both parieto-temporals, occipital and basilar regions.

Frontal region.—In the examination of the frontal region, we should endeavor to show on our Röntgenograms, as much of the vertical plate of the frontal bone as possible. This is best done by securely fastening, with adhesive strips or by other means an 8 by 10 inch plate or film—placed in black or orange envelopes—upon an inclined plane of about 25°, the center of the plate or film having been previously marked on the orange or outside envelope by means of a wax pencil (lead-pencil marks are reproduced on the negative). This inclined plane with plate attached is placed upon the top of a table, at the head, and securely fastened to same either by clamps or sandbags. The X-ray tube in a proper stand, is adjusted over the plate in such a manner that the vertical axis of a 2-inch opening in a lead diaphragm beneath the tube, representing the central ray, is centered upon the center of the plate or film at a distance of about 22 inches. A metal cone can be used if desired. The patient is then placed on the table, flat on the abdomen with head, face down, resting on the plate. The glabella should be in contact with plate or film at its central point. An imaginary line, running through the center of the forehead, glabella, tip of the nose and symphysis of the jaw, should rest upon the central line of the long axis of the plate. The head should then be firmly fixed either by clamps or by means of a 3-inch muslin bandage with small sandbags attached to each end, placed over the patient's head, the bags hanging on opposite sides of the table. Care should be taken not to rotate the head when the clamps are applied or the weighted bandage adjusted.

A 6-inch spark-gap is used, 40 to 45 milliamperes being put through the tube, and an exposure of about six seconds. In the majority of cases this procedure will give satisfactory Röntgenograms of the frontal region.

It is frequently the case that the patient's condition will not allow the manipulation necessary to place him in the above position. The procedure should then be reversed, the tube being placed beneath and the plate or film above.

Temporo-parietal region.—In examining the temporo-parietal region, we should show that portion of the skull lying between the sagittal suture above, the coronal suture anteriorly, the lambdoidal suture posteriorly and including the entire temporal bone. Both sides of the head must be examined.

A 10 by 12 inch plate or film, placed in a black and orange envelope, is securely fastened on the top of a 14 by 17 inch lead covered

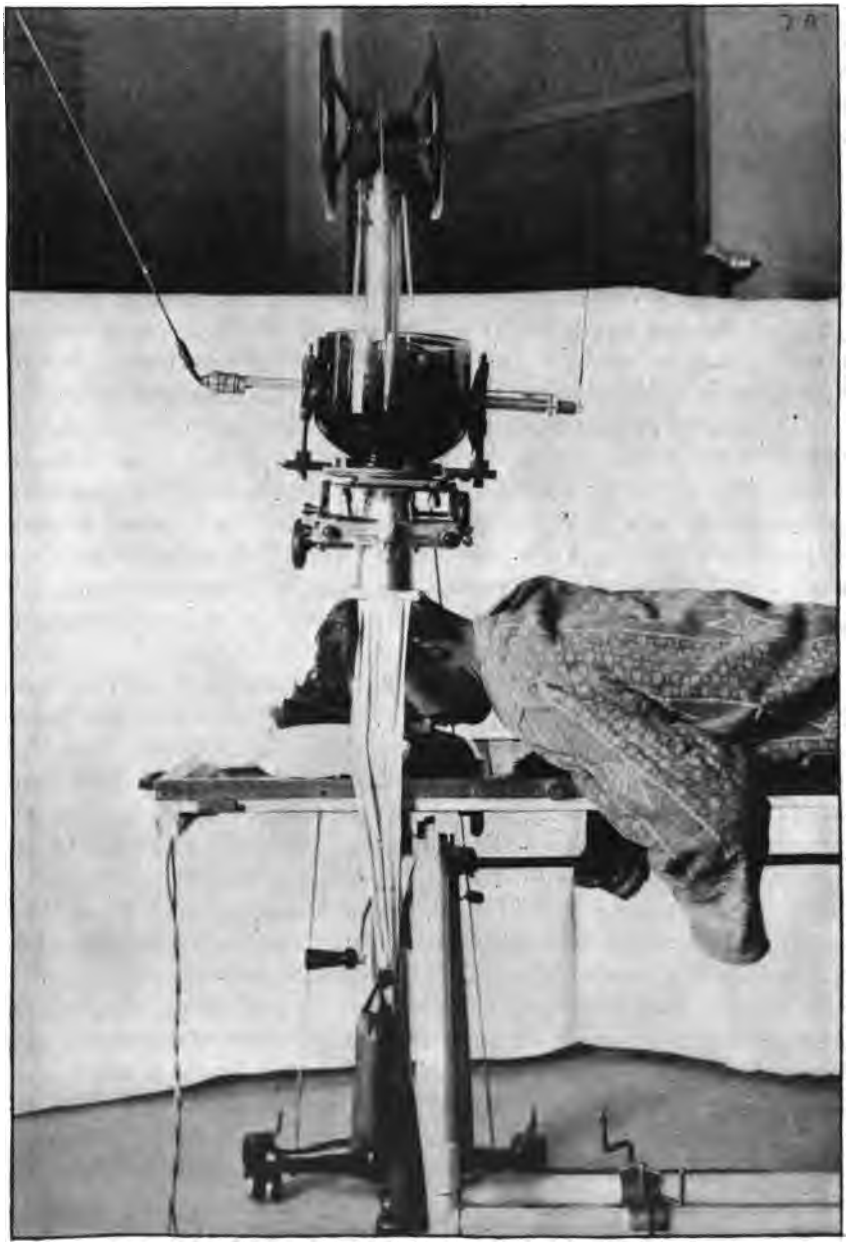

FIG. 70.

board, 1 inch in thickness, the center of the plate being plainly marked on the outside of the orange envelope. This lead covered board with plate attached is placed on top of three other wooden pieces of similar size and all placed upon the Röntgenographic table at the upper end. The patient lies on his side upon the top of the table, with neck slightly extended, the center of the temporal region resting upon the center of the plate, the base line being in contact with the long axis. The head is raised or lowered by adding or removing board units until a line, extending from the center of the forehead through the glabella, the center of the nose and ending at the symphysis of the lower jaw, is parellel with the plate. The boards should now be firmly clamped to the table or held in place by sandbags. A small platform, the top of which can be raised or lowered, may be used, if accessible, instead of the sections of board. The tube is adjusted over the plate so that the vertical axis of a 3-inch opening in a lead diaphragm beneath the tube is centered to the center of the plate, at a distance of 24 inches; this can be done by moving the patient's head to one side until the centering has been accomplished and then replacing the head in its original positon where it is securely clamped or held by the weighted bandage. The same electrical conditions govern the tube as when examining the frontal region except that it is seldom necessary to make the exposure longer than three or four seconds.

Occipital region.—When we examine the posterior region, we must bring out with clear detail the entire occipital bone, with the lambdoidal sutures like an inverted V above, the mastoids on either side and the foramen magnum below. To obtain this an 8 by 10 inch plate or film in envelopes is used, the outside one having been properly centered by ruling. This plate or film is securely fastened to the top of an inclined plane of 25°. The plane with plate attached is then placed upon the top of a table near the upper end, where it is clamped in position or held by sandbags. The X-ray tube is tilted 15° toward the foot of the table and adjusted over the plate so that the vertical axis of a 2-inch opening in a lead diaphragm placed beneath the tube, centers 1 inch above the center of the plate at a distance of 22 inches. The patient lies on his back on the top of the table with the occiput flat down on the plate, the center of the occipital bone being placed in contact with the center of the plate. The head should be slightly flexed so that the central ray enters the forehead just above and between the frontal eminences. If a cone is used, the lower border of the cone should not extend below the supra-orbital ridges. The head is clamped or held in this position by a weighted bandage. An exposure of about six seconds is made with a tube taking about 45 milliamperes and with a gap of 6 inches.

FIG. 71.

Basal fracture.—In cases of suspected fracture of the base we have been using, with some modification, the vertical technic as advocated by Dr. David R. Bowen, of Philadelphia, for examination of the sphenoidal sinuses as follows:

A small accessory stand, about the height of the Röntgenographic table, is placed at the head of the latter. A wooden board, covered with one-sixteenth inch lead on its upper surface, is placed on the top of this accessory table. Under the edge farthest away from the Röntgenographic table is placed an ordinary sandbag, thus making an adjustable inclined plane (a more elaborate specially devised, adjustable table top can be used if desired). An 8 by 10 inch plate or film, in black and orange envelopes, is placed on top, the lower edge of the plate being flush with the edge of the board nearest the long table. It is fixed with adhesive strips. The orange, or outside envelope holding the plate, is properly ruled and centered. The Röntgenographic table is now raised about 5 inches above the level of the accessory stand. The patient lies on his back on the top of the Röntgenographic table, the shoulders at the upper edge with a pad beneath and the head dropped over the end in such a manner that the top of the head rests upon the plate. The plane of the plate is now adjusted with the sandbag beneath the board until the level of the plate is parallel with a line drawn on the patient's face from the tip of the nose to the external auditory meatus; to accomplish this, it may be necessary in addition to raise or lower the Röntgenographic table, depending upon the length of the patient's neck and the obesity present. The tube is then adjusted, the vertical axis of a 2-inch opening in a lead diaphragm beneath the tube, being centered at a point midway between the mid-larynx and the symphysis of the jaw and at a point about 1 inch anterior to the external auditory meatus at a distance of about 24 inches; this is a variable angle so that in order to place the image upon the center of the plate we find it necessary to move the patient's head to one side and adjust the plate until the central ray is centered on the center of the plate; the head is then replaced in the original position. The central axis of the diaphragm opening should be perpendicular to the base line and the plate. The patient's head is firmly clamped or held in position by suspending the weighted muslin bandage under the symphysis of the jaw. A tube backing up about 6 inches and taking about 45 milliamperes is used; the time about 8 seconds.

On account of the exposure necessary to completely examine the skull it is advisable to filter the rays through one millimeter of aluminum. Care should also be taken to see that the focal spot on the target of the tube is properly centered to the center of the opening in the diaphragms.

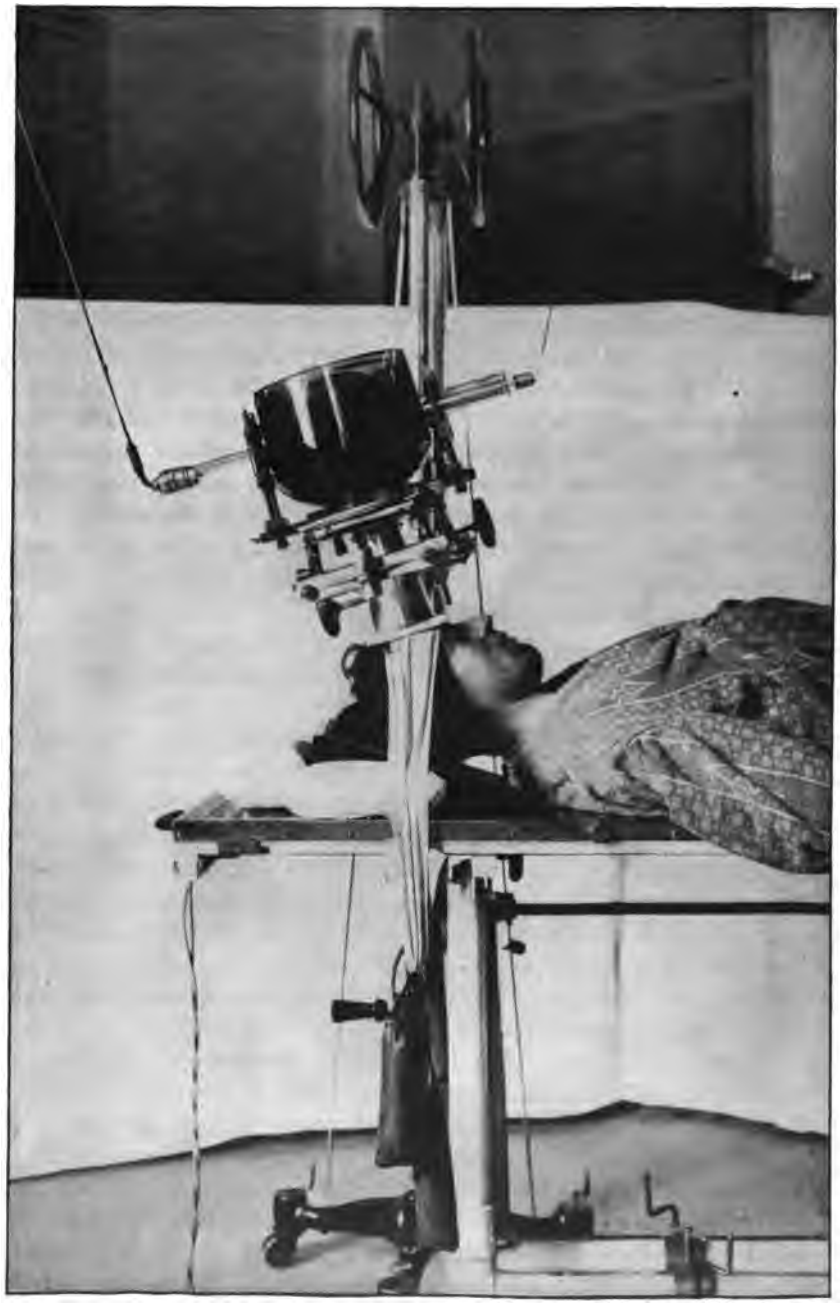

FIG. 72.

Having obtained satisfactory Röntgenograms, it is necessary to have the experience and anatomical knowledge to make the correct interpretation. In reading Röntgenograms of the base of the skull, beginning behind, we see the foramen magnum and within it the odontoid process of the axis. Just forward from this opening, on either side, the mastoids are distinctly reproduced with the associated shadows of the petrous portion of the temporal bone. Anterior is a clear view of the middle fossa. If we have not produced too much extension of the chin, the anterior fossa can be seen in front of the overlying shadow of the lower jaw. Fractures of the zygomatic arch, with the extent of displacement, are usually beautifully shown in Röntgenograms of the base.

In the interpretation of the frontal, lateral, and occipital regions one must remember the suture markings and the normal radiating lines cast by the grooves on the inner table of the skull which accommodate the meningeal blood vessels; these grooves spread fan-shaped from an anterior point backward. The shadows cast by the diploic spaces, between the inner and outer tables of the skull, are lines extending vertically upward from points at the base, directly in variance with the shadows representing the meningeal grooves.

Fractures on the Röntgenograms usually show as dark, sharply cut lines of varying width depending upon the extent of separation; they may be vertical, horizontal, or curved, but seldom directed in the same manner as the blood vessel grooves.

Stereo-Röntgenograms.—In addition, we wish to especially emphasize the value of the stereo-Röntgenographic examination in these head injuries. It is often difficult in cases of slight depression for the surgeon to decide whether to interfere or not. The presence or absence of symptoms of intracranial pressure and the actual demonstration of the condition of the inner table of the skull, with the amount of depression, if any, are vital facts which must be considered before the decision can be made.

Lateral stereo-Röntgenograms aid in tracing the fracture line into the base and locating the termination.

In the common penetrating fractures of the vertical plate of the frontal bone and those cases of linear fracture extending into the roof of the orbit, it is of extreme value as giving the most accurate conception of the seriousness of the bony injury.

If stereoscopic examination is to be made, a simple plate-changing device, such as recommended by Dr. Bowen, with a tube-stand so constructed as to allow a 6 cm. shift and a 10° tilt of the tube can be used; where possible, it is best to shift in the line of the long axis of the head.

FIG. 73.

CHAPTER IV.

INTRACRANIAL TRAUMA.

The brain is subject to the same types of injuries as other tissues. Such injuries may be localized, as those occurring in deformation of the skull at the point of trauma or from contusion of the brain against the unyielding walls of the skull and dura at more remote points.

A sudden impact from an instrument capable of rebounding will produce such a result. More widespread lesions, shown by scattered areas of contusion throughout the brain, result from the application to the skull of forces of greater mass and less velocity. In such ways the character of the injury modifies the pathological findings.

As the brain is of semi-fluid consistency and the integral parts of the central nervous system are not well supplied with supporting tissue, it is readily subject to injuries to its own structure and to cerebral vessels which have less support than vessels in other organs.

With localized injury, disorganization of the brain may result, with profuse hemorrhage into tissues which become separated. Such pulpified areas may be seen after a deeply depressed fracture or beneath the intact dura following tangential wounds of the skull. Widespread effects of brain injury are shown by small, scattered areas of vascular dilatation with small hemorrhages or minute thromboses in the cerebral vessels about which are patches of edema. If the rending quality of the force is sufficient, or if intracerebral hemorrhage is profuse, brain lacerations of various degrees will result. Examinations of brains after severe injury will show varying degrees of these changes.

The most common sites of gross injury seem to be the under surfaces of the frontal lobes and the tips of the temporal lobes. These facts must be kept in mind as a guide to future treatment.

The term "concussion" has in the past been intended to apply to the condition after head injuries in which profound symptoms may develop without any corresponding anatomical change in the brain. Unfortunately it has become associated in the minds of the profession as being that state in which unconsciousness with certain other symptoms may appear. In the light of more exact pathological studies it would seem safer to consider that the brain is subject to contusion of various degrees rather than to continue in the general

acceptance of a theoretical basis of functional disturbance which has resulted in loose conceptions of traumatic cerebral pathology.

After contusion the effects may be manifested by widespread punctate lesions or by localized gross changes. In the latter, hemorrhage may occur from the vessels of the pia-arachnoid, resulting in suffusion of blood between these two membranes. Such hemorrhages are usually associated with other lesions and are seldom great in degree. Similar hemorrhage may occur in the cortex of the brain from rupture of vessels.

While sub-cortical and intracerebral hemorrhages may occur, they are less frequent and usually complicated by surface manifestations. It is not infrequent to find the only gross changes in the tips of the temporal lobes and beneath the frontal lobes. The indirect force to which these parts may be subjected may even result in gross lacerations without any other findings at more remote points. In such instances and in many cases in which the force of the injury has exceeded the limits of cohesion of the brain, the appearance of parts of the brain may be that of a livid or plum-colored mass.

The immediate effects of injury differ from those appearing after twelve hours and continuing until organization takes place. After the first hours, edema ensues, which brings with it the added dangers of brain swelling and circulatory compression. While the subject of edema in all phases of brain disease is not thoroughly understood, the explanation of Cannon seems to make clear at least the edema following trauma. It will be recalled that tissues deprived of oxygen will take up fluid, and swell. With the rupture of many vessels of varying sizes and with thromboses in others, a considerable bulk of brain tissue may be deprived of its oxygen. The consequent edema, if the injury has affected a wide area, may be sufficient to raise the intracranial pressure to a degree beyond that which is within the power of the vagus and vasomotor mechanisms to meet.

Healing of a defect in the brain takes place partly by scar tissue formation, and partly later by the production of a rather broad zone of sclerotic neuroglia. Healing of the area of hemorrhage (intracerebral) takes place by the disintegration of blood and its gradual removal by phagocytes. Sometimes a cavity is left lined by granulation tissue which is deeply pigmented and contains a clear yellow fluid. The neuroglia may play a large part in forming the wall of this cavity. There is little tendency to the formation of granulation tissue.

Associated with brain injury, the vessels of the dura may also suffer. Hemorrhages from the middle meningeal artery or its branches and from the large venous sinuses as well, are common. Of the arteries, these usually result in extra-dural (epi-dural) hemorrhage, since they lie on the outer surface of the dura. In its course

from the base of the brain the middle meningeal artery grooves the base and vault of the skull, and occasionally may even tunnel through the bone for a short distance. It not infrequently happens that the bone may be sufficiently deformed by a compressing force to rupture this vessel in its groove and yet no fracture occur. It is of importance to remember that middle meningeal hemorrhage is not incompatible with an intact skull. Such hemorrhages usually are slow at first, as the dura, closely applied to the bone, strips with difficulty. As the hemorrhage increases and the dura pouches inward it strips off more rapidly. Such hemorrhages usually occur toward the lower part of the vault, and extend upward. In this location frequently the first objective signs to be noted in addition to those of general compression, are consequent upon involvement of the lower portion of the pre-frontal convolution. The motor distribution in this area has at its lowermost portion the area for representation of the face. This is usually first involved. With the upward extension of the hemorrhage, involvement of the arm, trunk, and leg of the opposite side of the body gradually appear. With the early onset of such hemorrhage it is of importance to differentiate carefully an isolated facial paralysis from such a cause from one resulting from injury to the facial nerve on the opposite side. Both of these conditions are rather common.

The classical picture of a hemorrhage from the middle meningeal artery is characterized by a period of consciousness (latent period) between the immediate effects of the injury and the later onset of stupor and unconsciousness. The latent period may vary from only a partial awakening of consciousness to a complete return of it and may vary in duration from a matter of minutes to a few hours.

It should be remembered that frequently the primary unconsciousness after injury may last a sufficient time for a slow hemorrhage to begin to manifest its symptoms without an interval or latent period.

In addition to large hemorrhages from one of the main trunks of the middle meningeal, separation of the dura from the vault is occasionally seen over a wide area with profuse hemorrhage. Such hemorrhages are probably due to a force bending inward the vault and disturbing its relation to the dura, followed by bleeding from a multitude of small vessels.

Certain of the large venous sinuses are exposed by their position to frequent injury, notably the longitudinal sinus most often involved by direct injury. During the present war, the frequency of this injury from tangential wounds has attracted especial attention to it and to the accompanying bilateral spastic paralysis. The associated bone and brain injury as well as the interference with venous return from both hemispheres to the sinus contributes to the resulting clinical picture.

The cavernous sinus being near the region to which all fractures of the base tend to converge, is peculiarly situated and readily exposed to injury. The petrous portion of the temporal bone, so commonly fractured, exposes the lateral sigmoid sinus also to these dangers. Other sinuses may be injured, but much less frequently.

Physiology of cerebral compression.—While in injuries of other parts of the body, the relief of tension from any cause is generally recognized as of importance, its prominence in head injuries deserves that it occupy the forefront of our attention. A clear understanding of the effects of cerebral compression is indispensable to the proper conduct of cranial injuries. Upon its recognition and the measures directed toward its relief the immediate outcome of the case may depend. In addition, the late troublesome sequelæ of brain injuries are favorably influenced.

A large amount of experimental work on the physiology of acute cerebral compression has given us a basis for the interpretation of our findings. Clinically, it must be remembered, however, that the reaction of the vasomotor and respiratory centers in an injured brain is not always as prompt as in the experimental cases in which compression was studied for the most part in normal brains. With this difference in mind, it is possible to explain satisfactorily certain reactions to compressing forces.

Kocher has divided the stages of compression into four. The first stage, in which the presence of the compressing agency is compensated for, at least in part, by forcing out of some of the intra-cranial fluid content normally present. This encroachment is largely at the expense of the cerebrospinal fluid content of the intra-cranial space. It is stated experimentally that about 6 per cent of this space may be encroached upon before the appearance of symptoms. With this relatively slight pressure some venous engorgement occurs. The first symptoms to appear are headache and slight mental dullness and possibly some focal symptoms.

The second stage is characterized physiologically by blanching of some of the cerebral veins and, clinically, by increasing headache, restlessness, irritability and intolerance of any interference, vertigo, and perhaps excitement or delirium. Focal manifestations depend on the location of brain involvement.

The third stage is that of manifest compression resulting from a degree of intra-cranial pressure which is interfering with medullary circulation. The deprivation of proper blood supply to the circulatory (vagus and vasomotor) and respiratory centers results in stimulation of the former. This stimulation results in a slowing of the pulse, constriction of the splanchnic vessels and consequent rise in blood pressure. The circulatory system then responds in such a way as to meet the demands upon it. During this stage, clinically, then,

there is slow pulse, later a rise in blood pressure, and deep stertorous respiration. To this picture is added whatever signs of localized brain injury may be present. In this state it may happen that the intra-cranial pressure rises to a degree to which it is not easy for the circulatory mechanism to respond. Under the stimulation from anemia of the medulla, the vagus will slow the pulse and the vasomotor centers react. When, however, this reaction occurs and the blood again reaches these centers, the necessary stimulation for the continuance of this reaction is lost and the blood pressure falls. In such instances, periodic fluctuations of blood pressure occur. Associated with these are respiratory changes. Periods of lessened respiratory action or of apnea may occur when the blood pressure is lowest. At such times other manifestations, as the widening of the pupils, may occur and the patient may pass into a most profound stupor and reflexes be lost. During the up-wave—that is, during the time of increasing blood pressure—stupor may be lessened, pupils contract, and the patient move or turn, reflexes return, and the respiration deepen. Such a response may ultimately become impossible with increased rises of intra-cranial pressure, and when this occurs circulatory failure becomes evident as in stage 4, in which there is a rapid pulse, blood pressure, which is decreasing, and deep stupor. This represents the terminal stage of compression. Relief of compression in this stage of failure of compensation is rarely of any avail.

Symptoms.—Signs of fracture of the skull and symptoms of brain injury, while independent, are so closely associated in the causes producing them, that one may be a guide in the diagnosis and treatment of the other. Symptoms of fracture are, however, discussed under that heading, and from a diagnosis of the site of greatest bone injury a clue is frequently given as to that place demanding most attention as regards the condition of the brain. Certain general symptoms in brain injuries form guides as to the character of the injury and are of aid in prognosis and treatment.

Temperature.—In general immediately following a head injury there is a lowering of temperature, which in the period of reaction is followed by an increase of temperature to or above normal. In cases progressing to a fatal termination the temperature usually shows a steady, gradual but irregular increase which at its height may reach 105 ° F. or even higher. The height of the temperature seems to be largely dependent upon the extent and gravity of the brain contusion. Rises of temperature above 104° F. are seldom seen in recovery cases. The degree of temperature reaction affords a measure of the amount of brain contusion. It is of importance that brain contusion rather than intra-cranial hemorrhage is the factor affecting the control of body temperature.

Involvement of any one particular part of the brain over any other seems to have little if any effect in producing this result. Explanation of temperature changes in association with changes in any single heat controlling center have not been supported.

Pulse rate.—Correlation of pulse rate with other physiological data is of greatest value. Of greatest significance is the slowing of the pulse, which may appear early in severe injuries if the patient is able to react. In a still larger group of injuries of less severity the pulse rate gradually slows in the first 12 hours and ordinarily reaches its lowest rate, perhaps as low as 55 per minute, where it remains for a considerable time. In the most severe cases, when first seen the patient may show an extremely rapid pulse which never slows and in which the case goes on to a rapidly fatal termination. In such, autopsy may reveal a condition which must certainly have caused compression of the brain, although no clinical signs of it appeared. The explanation for this is that the severity of the injury is sufficient to impair the physiological powers of reaction and the terminal stage (stage 4 of compression) was reached at once. In still other instances an initial rapid pulse may be succeeded by reaction on the part of the patient, manifested by rising temperature, slowing of the pulse, and perhaps a rise in blood pressure with other indications of pressure. It should be remembered that even after minor degrees of brain injury a slow pulse is common and frequently persistent. While a slow pulse necessitates that close observation be kept, it should not be considered, when no other signs are present, as a sufficient indication for the surgical relief of pressure. Such a slow pulse is frequently manifest as the only sign indicating a brain injury, and in such a case may not be associated with any rise in blood pressure. It may persist for long periods after convalescence is well established.

The pulse of compression is a slow, full volume pulse with increased tension. Without the appearance of other signs a slow pulse alone should not be taken necessarily to indicate a material increase in intra-cranial pressure. In association, however, with other signs, it is of the utmost value.

In brain injuries, rises in temperature are not accompanied by corresponding rise in the pulse rate. Asymmetry in the radial pulses, both in rate and volume, is occasionally found.

Blood pressure.—It is usual to find a blood pressure below normal if examination is made shortly after the receipt of the injury. In the cases which progress to a rapidly fatal termination, the blood pressure continues to fall. If, however, proper reaction occurs, blood pressure rises again to normal. Later on, the blood pressure will depend to a considerable degree upon the amount of intra-cranial pressure as outlined above. sual, however, to see high degrees of blood pressure pre re seen in certain chronic

conditions with hypertension. While experimental rise in blood pressure has been one of the most characteristic phenomena, it is not of equal value clinically. Considerable rises of intra-cranial pressure may be found before blood pressure changes begin. Absence of an increase in blood pressure is not incompatible with some rise of cerebral tension.

Respiration.—The shallow respiration seen at once following head injury gives place with reaction to deeper breathing, and with the onset of pressure the heavy stertorous respiration as seen in apoplectics is common. With increasing intra-cranial pressure, this type of breathing may change to a slower irregular or interrupted type. True Cheyne-Stokes respiration may occur, but is not common. Upon reaching the fourth stage of compression, respiration again becomes more shallow.

In that relatively small group of cases with direct medullary or vagus involvement, as from the pressure of a clot, certain respiratory phenomena may be extremely characteristic. Cyanosis, pulmonary edema, and respirations going down to a rate not oftener than two or four to the minute may be seen. The characteristic continuance of the heart beat for some time after complete respiratory failure occurs.

Pupils.—Of what value are the pupillary findings after head injury?

All conditions are met in a series of cases. Contracted pupils, greatly dilated pupils, equal and unequal pupils, reacting and immobile pupils, concentric and excentric ones. Not only do we see these varieties in a large number of cases, but in any case at different times we may find any or all of these occurring transiently. Their explanation is most difficult, and the pathological conditions governing them involve many factors. The brain lesions may cause either irritative or paralytic signs. The chief sympathetic trunks coursing with the internal carotids as they enter the skull are at the points to which the mechanical forces tend to converge. Injury to these trunks may stimulate or paralyze fibers concerned in dilatation of the pupils. The third nerve is rarely injured in its peripheral course. Its central connections are, however, subject to the same effects of contusion as are other parts of the brain.

There still remains a popular impression that the pupillary findings in head injuries are of much importance and have great significance. Usually they are the first findings to be made and commented upon. They hardly deserve so much note. From experimental and clinical work done, one seems justified in stating that with inequality of the pupils, in a little more than one-half of the cases the larger pupil is on the side of the greater intra-cranial pressure. The value

of this sign is so slight it should not be given too much weight when other less questionable signs are present. Dilated and non-reacting pupils have an unfavorable prognostic significance. The possibility of pre-existing pupillary changes, of course, should be kept in mind. While the condition of the pupils at any one time may be misleading, frequently repeated examinations and notes of the changes occurring in sequence may be of help in interpreting changing intra-cranial conditions.

Eye grounds.—Ophthalmoscopic findings are of very great value and are certainly among our most reliable signs in chronic intracranial pressure as represented in type by brain tumors. In acute cases as represented by the head injuries they are of relatively little help. In the first twenty-four hours even with marked intracranial pressure it is exceptional to find unmistakable evidence in the discs. After this time we secure more help from this examination, and the value of it increases with the lapse of time. Hemorrhages in the retina occasionally occur.

The tortuosity and fullness of the retinal veins, the hyperemia of the discs and beginning edema of the nasal halves help us materially in deciding our plan of treatment when they are present, and clear up for us the meaning of other signs which taken alone would be questionable.

Other symptoms which are frequently seen, as vomiting, lack of control of vesical and rectal sphincters, and chills, seem to have no relation to the site of the brain injury. No diagnostic or prognostic significance can be attached to them.

Spinal puncture.—Spinal puncture yields information of considerable value and occasionally is an aid in the treatment. It must be practiced, however, with some care, as not infrequently intracranial hemorrhage which has come to a stop may be started again by the lessening of cerebro-spinal pressure. Withdrawal of fluid must not be rapid for fear of herniation of the medulla and cerebellum into the foramen magnum. While this danger is much less than in neoplasms of the brain, all precautions should be taken to prevent it, and the stylet should not be completely withdrawn from the needle until the flow of fluid becomes a steady, slow, gradual one. In the event of intra-cranial hemorrhage the first fluid withdrawn by lumbar puncture may be entirely clear, owing to the fact that the blood has not yet diffused downward from above. A clear fluid removed soon after the injury does not rule out hemorrhage. Often with fluid continuing to flow it will change to one of faint pinkish tinge, and still later to a deep red fluid with great quantities of blood. Later punctures give us also some indications of the continuance of hemorrhage. The presence of a uniformly blood-stained

fluid is indicative of hemorrhage into the sub-arachnoid spaces or the ventricles. It may or may not be associated with signs of intracranial pressure or of localized brain injury. Repeated punctures for the relief of the edema following injuries are of some value. Measurements of spinal pressure should be made at each puncture (see Cerebro-spinal fluid, Chap. XI).

In cases which come to us many days after supposed head injury, the withdrawal of straw-colored fluid, even though red cells are no longer present, may give some indication of a previous injury.

Urine.—Examination of the urine shortly after receipt of the injury and again in the first 24 hours should be a routine. Too great significance should not be given to the finding of a few casts or a small amount of albumin, as these are not infrequent in cerebral lesions. The presence of sugar is of interest and of importance in showing the presumable course of the fracture. The convergence of the lines of fracture of the base of the skull to the sella turcica makes the pituitary gland occupy a somewhat precarious position. Just as in operative manipulation of this gland in animals, glycosuria follows, so also after fractures of the base which involve this area, glycosuria may be found in man. It is interesting to note in this connection reports of the occurrence of diabetes insipidus after head injuries.

Diagnosis.—In addition to general symptoms common in greater or less degree to all brain injuries, a detailed neurological examination in each case must add those symptoms appearing as a result of focal lesions and of cranial nerve involvement. Frequently, the inability of the patient to cooperate in sensory examinations limits us to our objective neurological findings. Particularly examinations of the special senses may be impossible for a period, but upon resumption of consciousness every effort should be made to secure this information at once in the light of possible subsequent happenings. Frequently repeated ophthalmoscopic examinations are essential. Slight differences in the reflexes or in the use of one or the other side of the body may even in the absence of compression symptoms give us a positive clue to a line of treatment before other findings make this apparent.

The signs of cranial nerve involvement and their relative frequency are dealt with elsewhere. The seventh and eighth nerves are commonly involved by fractures through the petrous bone. Involvement by hemorrhage is far more common than from direct implication in the fracture.

In a very small group the vagus is compressed, or the ninth, tenth, and eleventh nerves may be involved together.

The sixth nerve is occasionally injured as it grooves the lateral aspect of the dorsum sellæ.

The optic nerve is injured in 0.6 per cent (Phelps) by compression from the anterior clinoid process. The optic atrophy which is a primary one does not manifest itself objectively for about two weeks.

Unilateral or bilateral involvement of the first nerve is common. X-ray findings should be obtained at once following the neurological examination.

The signs of injury to the skull give indications of the location of the maximum brain injury in depressed and perforating fractures. This is less true of fissured fractures, although the location of these calls attention to the chance of injury to underlying structures; for example, in fracture of the squamous portion of the temporal bone possible injury to the middle meningeal artery or its branches is suggested, and bleeding from the ear attracts attention to possible involvement of the seventh and eighth nerves.

In diagnosis we desire to determine:

(1) The extent and degree of severity of the generalized brain contusion from a consideration of the type of injury and by the general signs.

(2) What are the conditions of intra-cranial tension?

(3) What evidence is there of localized brain involvement? In this, the results of the neurological examination—the presence of paralysis, and involvement of other specialized brain areas and a consideration of the bone injury (depressed and perforating fractures) are all concerned.

Localized brain involvement appearing within the first hours suggests localized hemorrhage, often of extra-dural origin.

Signs of general pressure appearing in the first twelve hours suggest free intra-dural hemorrhage. A localized brain lesion is usually accompanied by signs of general pressure.

After the first twelve or eighteen hours the appearance of general pressure is more apt to be the result of edema.

It is of the utmost importance to secure half hourly records of pulse, temperature, and respiration. Routine orders should be kept to this effect. The neurological, ophthalmoscopic, and blood pressure findings should be frequently repeated by the surgical attendant.

Treatment.—Isolation, quiet, sufficient liquids, attention to bowels and avoidance of over filling the bladder, ice packs to the head, are routine.

In those with extreme delirium, restraining sheets may be necessary. Any other method of restraint requires the closest attention to prevent injury. Spinal puncture may be beneficial. The use of bromides and chloral hydrate is seldom of any avail, even in large doses. Morphin is of greater help, but should be given only when rest is essential, and should be combined with atropin. Urotropin, 4 grams daily, is given in view of the possibilities of urinary tract

and cerebrospinal infection in mind, although in the latter case its value is questioned. Urinary acidity should be maintained during its use.

Depressed and perforating fractures require surgical treatment. Without treatment, cysts, focal epilepsy, adhesions and troublesome local symptoms follow the former, and in the latter is also added the danger of infection.

In fissured fractures the treatment is that necessitated by the associated brain injury and its complications. In fissured fractures the bone lesion in itself gives little concern. The gravity of the picture is determined by the brain and cranial nerve injury, the degree of intracranial pressure and the dangers of infection.

In the presence of signs of increased intracranial pressure if moderate or increasing, a sub-temporal decompression, as practiced by Cushing, with drainage for 48 hours, is advisable.

The operation is upon the side toward which the signs of greater injury point. With general pressure and no other determining factors, the right side is chosen in right-handed people.

Bilateral openings are frequently necessary. A drain of gutta percha tissue or a strip of rubber dam is placed between the temporal lobe and the base. The drainage of blood-stained cerebro-spinal fluid is often most profuse. Such a drain should be removed in 48 hours. A simple decompression without drainage is of little value. In this group of cases the intracranial pressure is due to increased fluid content within the skull. A decompression without drainage merely increases the cranial capacity, and shortly thereafter the original equilibrium is established between the general circulation on the one hand and the cranial fluid content on the other. With drainage of this fluid intracranial tension approaches normal, with consequent readjustment of vasomotor and circulatory control.

This procedure is advisable to relieve acute compression from either free hemorrhage or edema.

The presence of extra-dural extravasation is most frequent in the area exposed by the sub-temporal decompression. Bleeding from the middle meningeal may be controlled, and exploration toward the base or upward toward the motor cortex is easily effected. The drain is conducted through the temporal muscle and fascia. These prevent undue cerebral herniation and offer subsequent protection at the site of bone deficiency. The necessity for osteoplastic operations in this group of cases is rare.

Sub-temporal decompression with drainage usually answers all purposes.

After brain injuries prolonged mental and physical rest, with later carefully graded exercise and work adapted to the tolerance of

the individual, can not be too strongly emphasized. Complete abstinence from alcohol and avoidance of exposure to the sun and to heat is essential. Care should be taken to avoid accidents as a result of the dizziness and uncertainty which are such common sequelæ. A prolonged stay in bed after the subsidence of all symptoms and frequently a period of many months of inactivity after this time, offers most in the prevention of post-traumatic symptoms.

Prognosis.—The severity and location of the brain injury and the degree of compression are balanced against the patient's power of recuperation. The temperature curve is the most reliable index of the first. A temperature remaining sub-normal or a temperature rising to 102° F. and progressing steadily upward have a most unfavorable prognostic import. Conversely a temperature becoming normal and remaining so, or one rising to an upper limit of 102° F. but with no tendency to rise above, are favorable. If at the end of 48 hours the temperature is steady at or below 102° F. and the signs of compression are not increasing, prognosis becomes much more favorable.

Stage four of compression is to be regarded as a terminal condition.

Late effects.—These may be grouped as the results of injury to cranial nerves, injury to certain specialized brain areas, and the results of diffuse contusion.

Injury to cranial nerves.—Of these, permanent effects are seen often in the first, second, seventh, and eighth.

Anosmia—unilateral or bilateral—is common.

Loss of vision is infrequent (in less than 1 per cent) and is usually a result of compression of the optic nerve after fracture of the anterior clinoid process, less often from the result of hemorrhage into the sheath of the nerve. The atrophy is, of course, primary. Blindness is immediate, but the atrophy does not become evident for 10 days or two weeks.

Partial or temporary paralysis of the seventh is frequent. Permanent complete facial paralysis is rare. The former is due to compression by hemorrhage. A paralysis which is stationary is usually due to pressure from the fractured bone.

Deafness is common. The immediate impairment of hearing from hemorrhage into the middle ear is not to be confused with nerve injury.

Brain injuries resulting in hemiplegia, monoplegia, paraplegias, aphasias, defects in the visual field, etc., present the findings characteristic of lesions of the same parts from other causes.

Convulsive attacks may be characterized by focal manifestations or may be general. With focal or Jacksonian epilepsy, cortical

changes, scar, cyst, or adhesions may be found. Depressed bone may have been overlooked at the time of accident.

The large group of post-traumatic cases which without any focal manifestations present certain constant and general symptoms is of especial interest. Headache, dizziness, staggering, especially when lowering the head or looking upward as at the ceiling, extreme irritability, loss of memory, lack of power of concentration, inability to withstand the effects of heat or alcohol, and some general diminution of all special senses, are uniform and almost constant symptoms. These cases conform most closely to type. Objective signs are lacking, or perhaps there are unilateral sensory changes varying in degree. There is fatigue on the slightest mental effort or strain, and the mind becomes dazed, confused and bewildered. Self-confidence is lost.

In some, localized headache with tenderness may be accounted for by untreated depressed fractures or tender scars. In these, operative relief can be obtained. Chronic osteitis and meningeal adhesions are less frequent.

In a small number, signs of chronic intracranial pressure with moderate swelling of the optic disc, are found. These usually respond satisfactorily to decompression and drainage. In the others our greatest reliance is in preventive treatment. The medical treatment is symptomatic.

In prevention, the relief of high degrees of cerebral compression seems to lessen the incidence of post-traumatic symptoms, in addition to lessening the immediate mortality. The persistent impairment of physical and mental power so commonly seen makes it imperative that most minute attention be given to prevent its occurrence. The proportion of individuals who after head injuries have a lessened earning capacity or even permanent and total disability makes the problem of great economic importance.

CHAPTER V.

GUNSHOT INJURIES OF THE HEAD.

In considering the surgical aspects of gunshot wounds, one must keep in mind the lesions with which one has to deal. The term "gunshot injuries" is a very comprehensive one and includes not only bullet wounds and wounds of shell fragments to scalp, to skull, or to brain, but also the fractures of skull and trauma to brain that may accompany explosions of shells. Fractures of the skull, contusion, laceration of the brain, and hemorrhage, together with the resulting local or general compression, have been briefly reviewed in the preceding section. There remain for consideration, therefore, those injuries of the head in which the missile, bullet, or shell fragment, has injured the scalp, skull, or brain substance. While, of course, there are numerous combinations and variations, for the sake of convenience these injuries may be subdivided into the following groups:

I. Injuries of scalp and various types of cranial fracture, including tangential wounds. **The dura remains intact.**

II. The dura has been penetrated, with or without superficial injury to the brain, by bullet, shell fragment, bone, or clothing.

III. Subcortical injuries, with lodgment of foreign bodies deep in the substance of the brain.

IV. Perforating or through-and-through wounds of the cranium, usually bullet wounds.

V. Occlusion of the superior longitudinal sinus by a depressed fracture.

I. Wounds not involving dura.—The prognosis for these cases is on the whole good and the symptoms are not alarming, as a rule. There may be only a tangential wound of the scalp, (1) without a fracture of the outer table, (2) with grooving of the outer table, and (3) with or without fracture and comminution of the inner table, and meningeal hemorrhage. In a series of 33 cases of gunshot wounds of the scalp 29 were found afterwards to have fractures of the skull. In addition to the scalp and skull injury, though the dura be intact, there may be a subdural hemorrhage and a more or less serious contusion of the underlying cortex. According to the velocity and nature of the missile there will be greater or less injury to the brain, and the clinical picture will vary accordingly, typifying

that of contusion or hemorrhage with signs of local or general compression. One never can tell, however, how much damage to the cortex there may be when there is a depressed fracture of the inner table. With the dura intact there is not infrequently a localized contusion of the underlying brain. This area is more or less cone-shaped, with its base corresponding to the site of the depressed fragments of the inner table. The contused area comprises a mass of pulpified brain tissue mixed with blood, which is a potential source of complications. The pulped mass may cause disturbance of the local circulation; it may act as a source of irritation, similar to that of a foreign body; it is susceptible to infection and may resolve itself into a localized abscess or lead to meningitis or encephalitis; in the process of repair, it may be replaced by fibrous tissue and thus be converted into a remote source of irritation. After the skull has been opened and the dura exposed, this area of disintegrated brain tissues may be recognized by the discoloration of the dura, by the absence of free pulsation of the brain and by the doughy rather than semifluctuating sense to touch.

In the management of these cases, the wound should be excised. If there be a wound of entrance and exit and a bridge of tissue between, both wounds and the bullet tract itself should be excised and the surface of the skull exposed. When there is a depressed or linear fracture, or even when the pericranium has been torn, when the history and symptoms point to an injury to the cortex, sufficient bone should be removed for exploratory purposes. A small opening may first be made with drill or trephine through which the condition of the internal table may be seen. A depressed fragment should be removed for exploration, and the opening enlarged by nibbling the bone away with small forceps.

If the naked eye findings suggest disintegration of brain tissue or hemorrhage beneath the dura, even without the evidence of a focal lesion, the dura should be opened, preferably with a crucial incision, and the pulped brain tissue and débris removed. The dural wound is loosely sutured and a small drain of rubber tissue introduced through the scalp incision down to, but not beneath, the dura.

II. **Wounds involving dura.**—In wounds involving the dura, with or without lodgment of foreign material in the cortex, the prognosis becomes at once more grave. Fragments of bone, particles of clothing and hair, in addition to the missile, may be lodged in or near the surface of the brain, and apart from the damage inflicted upon the brain tissue there is the element of infection to be taken into account, with the probability, unless properly dealt with, of meningitis and encephalitis. As a matter of fact, death in most instances is due to infection of the damaged brain tissue, and unless this fundamental truth is recognized and an early and thorough operation conducted,

the mortality is necessarily high. This element of sepsis is the unanswerable argument for early operative interference, and as a corollary to this, one may add, that the fundamental principle of all operations is the complete excision of all infected parts and of all foreign material. The indications for operation in wounds involving the dura should admit of no discussion. The scalp wound should be excised after enlarging the wound when necessary. The bony opening should be enlarged with forceps sufficiently to expose one-third of an inch of uninjured dura. The ragged margin of the dural wound should now be trimmed away and the subcortical lesion dealt with according to the condition found. If time affords, the location and number of fragments may be determined by the X-ray beforehand. Without an X-ray one may determine whether any bone fragments have been in-driven by trying to piece together the fragments of the inner table as they lie on the surface.

III. **Sub-cortical injuries with lodgment of foreign bodies.**—The recognition of Group III, where the missile or foreign material finds lodgment deep in the substance of the brain, as distinct from Group II, where the lesion is more superficial, finds justification only in the fact that the injury is essentially more serious, and the question arises as to whether in all cases an attempt should be made to remove the foreign body. This group comprises at once the gravest cases of gunshot injuries and at the same time the most difficult of management. There is no doubt that in some cases retained fragments of bone or metal may become encapsulated and the patient recover and continue without symptoms.

Of 65 cases with retained missiles examined after evacuation to England, Sargent reports 30 per cent as completely recovered, 40 per cent as remarkably improved with relation to their neurological symptoms, and in only 10 per cent was there marked disability. From their observations, Sargent and Holmes draw the conclusion that it is extremely doubtful if surgical intervention, other than that necessary for the drainage and healing of the wounds, diminishes appreciably the risk of later complications, or can modify, except in a harmful direction, the course of these cases, from the functional standpoint. According to their records, many patients, with foreign bodies lodged deeply in the brain, recover and are scarcely more liable to serious complications than men in whom the brain has been merely exposed and lacerated. Any attempt to remove them, which may involve spread of infection or further destruction of brain tissue, is consequently inadvisable.

Contrary to these views, many surgeons still advocate attempts at removal of the foreign body at as early a date as possible, on the grounds chiefly that the foreign body is in most instances a potential source of infection. It should be remembered that the conclusions

of Sargent and Holmes are based not on all cases of penetrating wounds but only those which survived the immediate effects of the injury and were transferred to England.

There is, however, this generally accepted proviso that the removal of deep-seated foreign bodies should imply a careful preparatory X-ray and neurological examination, every facility for the localization and removal of the foreign body without the necessity of hasty action, a technic that takes into consideration the many intricate problems, and an operator trained in the peculiar difficulties of these war-bred intracranial procedures. Be it remembered, however, in dealing with these injuries, that the removal of the foreign body is not the only problem involved. Along the tract of the missile there will be greater or less laceration or pulpification of the brain. This detritus of macerated tissue, blood clot, hair, and what not, is a likely medium of bacterial activity, and its removal is as much a requisite of the prevention of infection and its sequelæ as is the extraction of the indriven bone or metal fragment. The invasion of the ventricles in these injuries adds measurably to the gravity of the prognosis. If not removed, foreign bodies may exert a deleterious effect upon the brain in one of several ways: (1) By acting as the carrier of infection, (2) by acting as a source of irritation to the brain tissue, (3) by interfering with the circulation of the brain, and (4) even after encapsulation, in the absence of infection, by giving rise to functional disturbance of that portion of the brain in which it may be located. The early removal of foreign bodies, therefore, not only inhibits infection, but facilitates the repair of the damaged brain tissue, favors an early and more complete restoration of function, and, in large measure, prevents serious sequelæ.

IV. **Perforation or through-and-through wounds of the cranium, usually bullet wounds.**—Most of these cases are killed outright. Those who survive do surprisingly well, and when the wound is by bullet there may be healing without infection. Both the wound of entrance and of exit need attention; the edges of the scalp wounds are excised, accessible splinters of bone removed, small drains inserted to the level of the dura, and the wounds partially sutured. Barany, however, believes that these wounds are surgically sterile during the first twenty-four hours and that primary suture without drainage will prevent invasion of infection from without.

V. **Occlusion of superior longitudinal sinus.**—The venous circulation of the surface of the brain is divided into two main parts; the superior veins drain the mesial aspect and upper half of the lateral aspect and empty into the superior longitudinal sinus; the inferior veins drain the lower half of the lateral surface and empty directly or indirectly into the cavernous sinous.

The symptom complex of thrombosis of the superior longitudinal sinus, first described by Holmes and Sargent, is dependent on interference with the superior set of cerebral veins, more particularly those which drain the pre- and post-central gyri, both motor and sensory areas.

The most frequent cause of thrombosis of the superior longitudinal sinus is a tangential wound of the vertex, crossing the mid-line somewhere near the upper Rolandic area (about 1 cm. back of the midpoint of the naso-inion line). The wound may be accompanied by depression of the whole bone, or of the inner table alone; or the sinus may be torn by spicules of bone, or directly damaged by the projectile. The sinus thrombosis may be complicated by wounds of the brain substance.

The characteristic features of the clinical picture are:

(1) The distribution of the motor symptoms.

(2) The early rigidity.

(3) The character of the sensory disturbance.

(1) *The distribution of the motor symptoms.*—The maximal circulatory disturbance will affect the mesial surface (paracentral lobule) and upper half of the Rolandic region, diminishing toward the mid-Rolandic region. According to the laws of cortical motor representation, then, we shall have affected in order of severity, toes, ankle, knee, hip, shoulder, elbow, and wrist. The wrist will be little affected, the fingers not at all; and the face which is represented still lower in the motor cortex, will also escape. The paralysis is always more marked in the leg than in the arm, and if equally severe in the two at first, soon begins to clear up in the distal arm segments. This distribution of paralysis differs from that found in the ordinary types of hemiplegia, where the arm is usually more affected in the distal than the proximal segments.

(2) *The early rigidity.*—Within 24 hours or less there is marked rigidity (spasticity) of the paralyzed parts. It may be so extreme that passive movement is impossible. Within from 24 to 48 hours it reaches its maximum, and shows no further tendency to increase. On the other hand, it improves as the paralysis improves. Even where it persists for a long time, there seems to be no tendency to the formation of organic contractures. Along with the early spasticity go increased tendon jerks and the Babinski reflex. This early rigidity is in marked contrast to the usual findings in hemiplegia. Ordinarily there is flaccidity of the affected limbs, with loss of reflexes for at least several days following the stroke. The same is true of the majority of cranial wounds other than the type here described.

(3) *Sensory disturbances.*—These are of the cortical type. Pain and temperature are unaffected. Touch is little affected. The chief

disturbance is in the sense of position and passive motion, localization of stimuli, and discrimination of compass points.

In general there are evidences of marked increase in intra-cranial tension—severe headaches and considerable swelling of the optic discs, more than the blurring of the margins commonly found in cranial injuries.

Certain other symptoms may occur, as transient bladder disturbances, paralysis of associated ocular movements, and even paralysis of the oculomotor or trochlear nerve.

Depending on the extent and location of the wound, all four limbs may be involved, or both legs and one arm, or in unilateral injuries a hemiplegic type may occur. Where the lesion lies well back, 5–6 cm. posterior to the midpoint of the naso-inionic line, there may be no paralysis or increase in reflexes, but only sensory disturbances of the type described above.

For some as yet unexplained reason the mortality following operation on these cases would seem to make it prohibitive. Makins' series showed a mortality of 38 per cent among 39 operated cases and half of 1 per cent among 37 unoperated cases. However, in this series only the very severe cases were operated upon.

General operative considerations.—The most momentous question from the standpoint of mortality is as to when and where the operation should be performed. Obviously an intracranial procedure, such as that involved in the removal of deep-seated foreign bodies, could be performed only at the evacuation hospital or the base hospital, either within a few hours or a few days of the injury. There are those who advocate postponement of the operation for two to five days to enable the patient to recover from shock, to permit of the formation of adhesions between brain and meninges, and to avoid the necessity of early transportation after the operation. Were this program adopted, the operation would be deferred until the patient reached the base hospital. There is a growing conviction, however, in opposition to this conservative line of thought, that more lives can be saved by early operation—operation within a few hours of the injury—performed at the evacuation hospital. Unless military exigencies will permit of head cases being retained near the front for operation and for a fairly long after-treatment, it is not likely that the mortality and loss of function will be materially reduced. Even though the practice of routine operations should include a number of minor injuries, no harm is done and the soldier will be able to return to duty at a much earlier period (Gray). On the other hand, there are many objections to the policy of delay. In the first place, the opportunity for removing all infective débris before the process of infection becomes established and spreads to adjacent tissue is lost

if the operation be delayed. The incidence of encephalitis and meningitis, the factors determining the life or death of the patient, will be the greater the longer the operation is deferred. The reactionary edema consequent upon the injury tends to close the tracts leading to the indriven projectile and varying degrees of prolapse with clots and damaged brain tissue will take place. Consequently, there is the greater difficulty of reaching the foreign body and the greater likelihood of lacerating the surrounding brain tissue in attempts at removal. The conclusion follows, therefore, that given the opportunity for the preparatory examinations, neurological and X-ray, and the proper facility for the conduct of the operation by those familiar with the problems involved, the sooner the operation is performed the lower will be the mortality. Per contra, the mortality of secondary operations at the base hospital following incomplete operations nearer the front has been exceedingly high.

In discussing the time and place for operation a word should be said of the effect of transportation. It has been an almost universal observation that the transportation of patients within a few days of the operation seriously affects the chances of recovery. The percentage of fatalities is lower the longer the interval between the operation and the time of evacuation. While there are occasions when military necessity makes early evacuation imperative, the patient should not be moved until two or three weeks have elapsed.

Preparation of the field of operation.—If not entirely shaved the scalp should be shaved at least over an area wide of the wound, as one can not tell how long incisions may be required before the operation is begun. The scalp should be cleansed with ether, benzin, or alcohol and the surface painted afterwards with tincture of iodin.

The anesthetic.—Whenever possible the operation should be performed under local anesthesia. It is, of course, not feasible when the patient is restless or uncontrollable. One hour before the operation a hypodermic of morphin, gr. one-sixth, or omnopen, gr. one-third, should be given, and repeated if necessary. The most dependable agent is procain, 1 per cent, to which should be added adrenalin in the proportion of 15 drops to 30 c. c. This solution not only renders the line of incision painless, but controls to a considerable degree hemorrhage from the scalp. When the patient is unconscious and in profound stupor no anesthetic will be required, while for the restless and uncontrollable patient a general narcotic, preferably ether, should be employed.

Incision.—One must choose between a flap exposure and a tripod or three-legged (Isle of Man) incision. The former is suitable for injuries in the occipital or temporal region, the latter for injuries of the vault. In both instances, however, the margins of the gunshot

wound should be completely excised. The tripod incision permits of a wide exposure of the skull, and by undermining the three flaps approximation is not difficult even after the contused margins of the missile wounds have been excised.

Management of skull injuries.—In the tangential wound with grooving of the outer table, one must be governed by the clinical picture and by the X-ray as to whether the skull should be trephined. When there is evidence of injury beneath the dura or when the X-ray shows comminution or depression of the internal table, the skull should be opened for purposes of exploration, and after the exposure of the underlying dura the question will arise as to whether the dura should be opened. When the dura is tense and discolored, when a doughy sensation is communicated to the examining finger in the absence of pulsation, and when there is clinical evidence of a cortical lesion, the dura may be opened providing, of course, one has been scrupulously careful to avoid infection.

When the foreign body has penetrated the skull, or when there is a mass of pulped brain tissue beneath the dura, obviously sufficient bone must be removed to permit of the necessary exploration. For this purpose one of two procedures may be adopted, the more common one of enlarging the opening with rongeur forceps or the removal of a section of bone *en bloc*. To accomplish this the skull is opened at four points of a rectangular space, including the wound of entrance, with Hudson or Doyen burr, and the intervening bone sectioned with De Vilbiss or Montenovesi forceps.

Removal of foreign material.—The X-ray will have determined the number and location of the metal or bone fragments. If superficially situated they may readily be detected with the finger and removed. The brain detritus, together with such foreign material as may have been carried in with the bone or missile, should be washed away with saline solution. The dural incision and scalp wound are closed with allowance for the introduction of tubular or rubber tissue drainage.

Removal of deep seated foreign bodies.—We have advocated, with certain reservations, the removal of all foreign material at the earliest possible moment, that is, at the evacuation hospital two to six hours after the injury. A prerequisite of course is accurate X-ray localization both as to number and locality. Prior to any attempt at removal, however, the tract should be thoroughly cleansed of brain pulp, blood clot, and presumably infected tissue in the manner prescribed in the next paragraph. The removal of the foreign body with the aid of a fluoroscope is, of course, the ideal method but frequently is not feasible. To push the finger no matter how gently into a tract tends to crowd the detritus of the wound into the surrounding healthy brain

tissue. A better method is the use of a catheter to determine the depth of the foreign body. The removal of metallic foreign substance should be attempted by way of the existing tract. Much unnecessary and irremediable damage has been done by the indiscriminate use of the magnet, but with practice and due consideration for the localization of important tracts and centers the brain may be spared much insult. A wire nail with rounded point is inserted gently down to the metallic fragment and attached to the electro-magnet. In non-magnetic objects one must rely upon the use of some form of extracting forceps. These instructions for the removal of foreign bodies are given with the understanding that they shall be applied with discretionary judgment. There will of necessity be circumstances and conditions to which attempts at removal would not be countenanced.

Toilet of the wounded brain.—The brain may be injured in one of two ways. Either by depressed fragments of bone, as in fractures, or by in-driven fragments of bone or missile. Even in the tangential wounds of scalp or skull there may be an extensive fracture of the internal table and the distance to which the inner table is bent determines the extent of damage to the brain. But with dura still intact there will be found often more or less extensive areas of damage to the cortex, perhaps, extending cone-fashion a distance of 1 or 2 inches into the brain. Here will be found a mixture of pulpified brain and blood clot which, if undisturbed, often becomes infected. Through a small incision in the dura this material will well out "like grease from a collapsible tube," tension is relieved and pulsation returns. The dural wound is closed and a small drain introduced down to the dural wound. When normal brain tissue follows the detritus this indicates excessive intracranial pressure and calls for a lumbar puncture. When the brain has been damaged by the passage of a solid fragment of bone or metal, tracts are established which are filled with blood clot and brain detritus. These tracts must be cleansed if infection is to be eliminated. The lesions are chiefly mechanical and microbic and must be treated by mechanical and antimicrobic remedies. Two remedies have been used for the removal of the material—saline irrigation and suction. The latter is the more convenient and the safer. A soft rubber catheter is attached to a Carrel-Gentile glass syringe and with gentle suction the brain pulp and small fragments will be evacuated until the tract is free. Normal brain tissue will not be drawn into the tube by the degree of suction of the average rubber bulb. Thus the brain is relieved of the greatest potential source of infection.

Drainage.—As a general principle one should avoid subdural drainage, whenever possible. This rule does not apply to the small rubber tube or strip of rubber tissue which one may insert at the angle

of a scalp wound. The principle indications for deep drainage are the presence of pus, of infected foreign bodies, of infected blood clot, or of continuous oozing from the damaged brain tissue. When the foreign and the pulped brain matter have been removed a small rubber tube or rubber wick may be inserted through the angle of the wound to, but not into, the opening in the dura and retained for 24 hours. On the other hand, when there is marked and continuous oozing, when pus has already formed, when a blood clot has become infected or when the foreign body is still retained, drainage is imperative and must be continued a longer or shorter time according to the nature of infection. In the presence of visible pus bacteriological examinations should be made and if streptococci be present, drainage should be continued until they disappear.

"It should be remembered that foreign bodies, especially flat pieces of bone, compress the brain in front of them; therefore, although they may be found at a depth of, say, one and a half to two inches, it is not necessary to push a drain in to that depth. The distal end of the track will, after extraction of the foreign body, be found to have approached considerably nearer the surface of the brain. If one attempts in such a case to push a stiff drain in for a couple of inches, there is a great likelihood that the lateral ventricle will be perforated by it. Immediately before inserting the drain, one should gently explore the track with the finger or catheter, and push the drain in only so far that it will not quite reach the extremity of the track. As a rule, the drain should be shortened slightly every day or every second day unless pus continues to discharge from the depth in fair quantity. It should be borne in mind that a drain, especially a rigid one, acts like any other foreign body, and may stimulate pus formation, besides providing a channel for possible entrance of fresh infection. All drains should on this account be removed as early as possible. In most cases they can be taken out on the second day.

"Rigid drains are harmful to the brain, especially those with holes cut in them. The intracranial pressure may force normal brain through the holes or end of the tube. The constant friction of the pulsating brain against a hard foreign body must have a bad effect. The most satisfactory drain, on the whole, seems to be a piece of jaconet, batiste, or similar substance, folded concertinawise. No apparent harm has followed the insertion, on the point of the finger, of a small amount of a paraffin paste 'bipp', flavine, brilliant green, or chloramine-T, etc. In certain cases, where the pus is particularly thick or profuse or where streptococcal infection is present, it may be advisable to insert in addition one or two narrow tubes. Antiseptic solution or thin paste may be instilled gently, a few drops at a time, every few hours. One must be careful, when inserting a

drain, that damage is not done to the healthy brain lining the track. One must therefore note the direction of the track very carefully." (Gray.)

Contrary to the experience of Gray regarding the use of solid tubes, Sargent and Holmes have found fenestrated cylinders of zinc, aluminum or copper, 1¾ inches long and ⅜ inch in diameter, very serviceable. A tube of this sort they bring out through the original wound and insert rolled rubber sheeting drains in the lower angles of the flap. Filling of the tubes with extruded brain matter is almost inevitable. Before completion of the operation this is curetted out, the lumen dried and filled with sterilized glycerin, and gauze soaked in glycerin is placed around the tube. Glycerin being hypertonic, inhibits bacterial growth and "seems to emulsify and facilitate the escape of the disintegrated brain substance." The procedure of curetting and syringing out the contents of the tube, drying, and refilling with glycerin is repeated daily. The tube is allowed to remain for about a week and is rotated daily to free the holes in the tube of brain matter and granulations.

Dura.—A word might be said of the management of the dura, when it is to be opened and when not, when closed and when not. Purposeful opening of the dura should not be undertaken if there is any doubt as to one's technic or as to the cleanliness of the surrounding field. Should there be any doubt, much better leave the dura alone, even though at a later date it may be necessary to operate for the evacuation of a disintegrating blood clot or a circumscribed abscess. By that time the region above and below the dura will be well walled off. But when one is confident of his surgical team and equipment the purposeful opening of the dura is quite justifiable, in fact, clearly indicated when (*a*) the dura is tense and pulsation lost (to the examining hand the underlying clot and brain pulp gives a doughy feel), (*b*) when there are clinical signs of serious functional disturbance. After the toilet of the contused area the dural wound made in crucial fashion is closed lightly and a drain introduced down to but not through it.

When the dura has already been penetrated by splinters, bone or metal, the dural wound should be treated like the scalp wound, that is, as though it were infected. The margins of the dural wound are excised with care so that surrounding adhesions may not be unnecessarily disturbed.

Use of antiseptics.—Cushing maintains that the use of watery solutions of antiseptics in the brain is objectionable on the ground that they lead to edema and a greater tendency to hernia. He has found the oily preparation Dichloramin-T really efficacious and believes it has enabled him to close, without drainage and without

harmful sequelæ, wounds which otherwise would have become infected. At the final stage of the wound toilet a small amount of Dichloramin-T (oily solution) is injected along the course of the tract through a small catheter and, just before final closure of the scalp, the dural surface of the wound is moistened with the same solution.

Hemorrhage.—The general principles affecting the control of intracranial hemorrhage as practiced in civil life have no special application in military practice. The adrenalin content of the local anesthetic reduces the scalp hemorrhage to a minimum; bleeding from the middle meningeal branches is readily controlled by ligature, rarely may it be necessary to ligate the external carotid artery; bleeding from the brain surface from the pial vessels is most easily and effectively checked with a muscle or fascia graft. These never fail. In military practice the injury to sinuses is of course relatively more frequent. A bullet, shell fragment, or sliver of bone readily penetrates the sinuses and because of the more exposed position, the longitudinal sinus is the most frequently damaged. For bleeding from this source sometimes a silk ligature will insure control, but, should the wound be more extensive, the so-called "postage-stamp" method is the method of choice. Upon exposure of the wound and removal of the foreign body, the hemorrhage is at once controlled with the tip of one index finger. On the tip of the index finger of the other hand a small fascial graft, removed from some other part of the body, is stuck and as one finger is withdrawn the "stamped" finger replaces it. Firm pressure is made for a few seconds, and when finger and pressure are removed hemorrhage is under control.

The use of the heavy rubber tourniquet to control hemorrhage from the scalp may be advisable in cases of profound shock, when the loss of a small amount of blood may count, but this can not be employed in the conscious patient and is therefore not applicable to those operated upon under local anesthesia.

Post-operative treatment.—The patient should be placed in the semi-sitting posture as soon as post-operative depression or shock has disappeared.

The possibility of infection will be a constant source of anxiety to the surgeon. A rise in temperature, an increase of headache, nausea or vomiting, muscular twitchings, delirium, stiffness of the neck, an increase of polymorphonuclear leucocytes in the cerebro-spinal fluid, together with signs of increasing pressure, will be evidence enough of meningitis or encephalitis. The wound must be opened, more thorough drainage established, and the sinus antiseptic ized with Dichloramin-T.

There may be a slight cerebral prolapse for a few days after operation, owing to the reactive edema. This will subside spontaneously, but to relieve undue pressure or discomfort, lumbar puncture should be employed.

Aspirin will usually relieve the post-operative discomfort. Urotropin, 2 to 3 grams daily, should be given from the day of injury and continued for several weeks in the more serious penetrating wounds (being comforted more perhaps by faith than by the inherent value of the drug); trional or morphin should be given freely to insure rest, quiet, and sleep in those who are nervous, restless, or delirious. Bromids throughout the convalescence, even for weeks, has the indorsement of some as a prophylactic against epilepsy. Transportation carries with it a distinct menace to the patient's welfare. It should not be ordered until two, preferably three, weeks have elapsed.

Results.—The mortality following gunshot injuries is of course in direct proportion to the depth and extent of the injury. The majority of patients with scalp wounds, with injuries to the skull with or without cortical contusion but without in-driven fragments, will recover in large numbers. The penetrating in-driven fragment carries with it, of course, the highest toll. While it is useless to quote figures, so widely at variance are the methods of computation, it may be said that the mortality is gradually going down from 50 per cent to 40 per cent, and under favorable circumstances even to 30 per cent. When the foreign body has penetrated a lateral ventricle the mortality is exceedingly high. Of this it is quite certain, however, that this lowering of mortality, this rescue of lives, has been made possible by only two elements: (1) The relegation as far as possible of head injuries to the surgeon especially trained and versed in the technic of the cranial surgery in war time; (2) the radical and thorough operation performed at the earliest possible moment, in contrast to the primary incomplete operation, with early evacuation to a base hospital. The motto for the surgeon undertaking these early operations should be "all or nothing."

Of the later results of gunshot injuries of the head the investigation of Sargent and Holmes of the condition of 1,239 patients from 2 to 18 months after the infection of the wound are of interest.

As to the mortality, only 46 or 0.37 per cent, died after admission to the London Hospital, and this number included nine who died within two weeks. In the large majority, in 41 out of 46 cases, death occurred within three months. The immediate cause of death in most instances was the spread of infection, although in a few death was due to intracranial hemorrhage.

TABLE OF THE CAUSES OF DEATH AFTER EVACUATION TO ENGLAND.

Intracranial hemorrhage	3
Following operation in England	11
Meningitis	10
Cerebral abscess	9

As to the physical disabilities, the amount of improvement of the paralysis, sensory and visual disturbances was surprising. This was attributed to the fact that in large measure these functional disturbances must have been due not to local destruction of brain tissue, but to contusion, edema, and to vascular disturbances. According to Sargent and Holmes, it may be expected that a considerable proportion of men with severe head injuries will be able to lead useful and active lives. Especially striking has been the improvement in the hemiplegias, diplegias or quadriplegias due to longitudinal sinus injuries.

The proportion of cases in which either insanity or epilepsy developed was surprisingly low. During a period of 12 months only 8 patients developed mental symptoms necessitating their admission to an insane hospital, and of these 4 had been discharged as cured or improved, 1 had been previously insane, and 1 had dementia precox. In only 2 cases could the persisting mental symptoms be attributed to the head injury.

What is true of the infrequency of insanity may be said also of epilepsy. Of 610 cases with complete records, 37, or 6 per cent, had had a convulsion and in only 11 of these were the convulsions frequent. In several the convulsions were arrested by bromides, and in 5 by secondary operations. In 33 of the 37 cases the injury to the brain was of a serious character; in 7 the lesion was a compound fracture with involvement both of dura and brain tissue.

Of the other neurological conditions there were, of course, various forms of paralysis, sensory and visual disturbances due to primary injury or subsequent complications, but in addition there was a large number of subjective disturbances not attributed to any local injury. They seemed to bear no relation at all either to the site or the severity of the injury. These subjective disturbances included symptoms usually associated with traumatic neurasthenia, such as headache, dizziness, emotional unrest and temperamental changes. There were only a few with major hysteria. It is interesting to note that these patients were incapacitated for active service.

Hernia cerebri and fungus cerebri.—Hernia cerebri is said to occur in one-half of all cases with loss of bone substance. It is more frequent in the parietal than in the frontal and occipital regions.

Of 610 cases of head injury returned to England 120, or about 20 per cent, had a cerebral hernia. Classified as follows (Sargent and Holmes):

Of 96 penetrating wounds with retained missiles there were 20 hernias, or 20.8 per cent.

Following removal of missile at base hospital or casualty clearing station (2 deaths)	6
Evacuated with missile retained (2 deaths)	14
Total	20

Of 68 cases of perforating wounds there were 14 cases of hernia, or 20.6 per cent.

Submitted to operation	10
Deaths	4
Wound completely healed	8

Of 310 cases of penetrating wounds without retained fragments 86 cases or 27.7 per cent.

Operated upon in France	62
Total deaths	19
Wounds completely healed	49

The prognosis depends entirely on the cause of the hernia and its susceptibility to treatment, upon the area of brain involved or upon the existence of an encephalitis or meningitis. The presence of pulsation is favorable; its absence may mean considerable displacement, stretching or rupturing of fibers adjacent to the protruding mass. As to prognosis and treatment, the exciting cause of the prolapse is of prime importance. This may be either (*a*) traumatic or (*b*) septic.

(*a*) The traumatic or non-septic hernia may be caused by increased intracranial pressure from hemorrhage, venous or cerebrospinal fluid engorgement (within or without the ventricles), a large area of cerebral contusion, and, further, the explosive effect of projectiles may cause immediate extrusion of brain substance from the osseous breach. Treatment of this group usually meets with definite response. The patient must be kept absolutely quiet, preferably sitting up. In the absence of infection the protrusion may recede in the course of a week. If not, lumbar puncture on alternate days, or daily, will frequently reduce the protrusion and relieve the usual symptoms of lethargy, headache, and mental irritability and the occasional attacks of Jacksonian epilepsy. On account of the danger of tearing protective adhesions between the brain and meninges by a too sudden recession of the hernia, the fluid should be allowed to escape from the needle only very slowly and not too much withdrawn at one sitting. If lumbar puncture fails and the hernia increases in size with evidence of alarming pressure, either local or general, a contralateral decompression is justifiable. All herniæ should be protected from possible accidental

injury by surrounding the dressings with a large buffer of wool. Morphin is to be given freely if the patient is restless or excited. The exposed surface requires daily dressing. Assuming the absence of infection, the surface may be dusted with boric acid or painted with tincture of iodin and a dry dressing applied. In the removal of dressings, the old dressing should be thoroughly moistened so that it can be removed without lacerating the cortex. In cases where the surface is covered with healthy granulation tissue the final closure of the wound may be expedited by skin grafting.

(*b*) Herniæ of septic origin instead of tending toward spontaneous recovery increase sometimes slowly, sometimes rapidly in size, and the greater the protruded mass the greater the interference with the venous circulation. Thus a vicious circle is established.

The nutrition of the protruded mass is seriously interfered with and softening occurs with the development of a true fungus cerebri. As the herniated mass increases in size it becomes so snugly engaged in the margins of the dura and bone that the drainage of infective material from beneath the dura is interfered with and an encephalitis or meningitis is a not uncommon sequel. The hernia may in some instances contain a diverticulum of a distended ventricle, and the persistence of a hernia should arouse one's suspicion as to the presence of an abscess.

Early operation and the prompt removal of potential sources of infection are truly prophylactic measures. If this practice is rigidly adhered to, septic herniæ with their train of disasters will be very much less frequent. As for the active treatment, it is needless to say that where there is evidence of strangulation this must be immediately relieved by enlarging the opening in bone and dura, and when there are grounds for suspecting abscess, as in the later stages, the abscess should be evacuated and drained. If the fundamental cause of the hernia is the encephalitis from retained foreign bodies and débris, these must be removed. To accomplish this it will be necessary, of course, to enlarge the bony opening, and after that the same principle should be observed as to the technic prescribed for the removal of foreign bodies. As in the non-septic variety, lumbar puncture may be required to relieve tension and later a contralateral decompression. The exposed surface of the herniated mass requires daily dressing. The surface may be cleansed with hydrogen peroxid and fomentations of saline solution applied. Instead of the latter the surface of the brain may be painted with tincture of iodin. Painting the surface with a 40 per cent solution of formalin every third day and 2 per cent solution of iodin in the two intervening days has been recommended by Rawling. In some instances as a last resort the protruding mass has been shaved off little by little,

and while at first sight this might seem unwarranted, inasmuch as the function of the protruded mass has by this time been destroyed, the final result as far as function is concerned, will be affected in no wise.

Meningitis.—All that has been said of the importance of removing the sources of infection might be appropriately repeated in discussing the treatment of meningitis. Prophylaxis must be the anchor-sheet, since after the process is once established and becomes diffuse, involving the base as well as the cortex and later the ventricles, surgical measures are of no avail. Among the preventive measures should be included the prompt removal of infective agents, adequate drainage, unnecessary enlargement of the dural wound, too rapid withdrawal of cerebrospinal fluid in septic hernias, and scrupulous care in the performance of operations to avoid possible sources of contamination. As for the active treatment once the diagnosis is established, we must depend upon lumbar puncture daily or oftener if need be. Attempts to establish permanent drainage either from the cisternae pontocerebellaris or from the dural sac in the lumbar region are not indorsed. There are, however, occasional exceptions.

Encephalitis.—Though clinically indistinguishable from meningitis, encephalitis is the more frequent and the more serious lesion of the two. The symptoms are due to increased intracranial tension as well as to the progressive destruction of brain tissue. The suppurative form is rapidly and progressively fatal. Brain tissue may be discharged from the wound, or fungus cerebri may develop and the process may terminate in abscess formation. The treatment involves the problem of the removal of all sources of infection, the establishment of adequate drainage, and the treatment of the accompanying hernia.

Abscess.—Abscess of the brain is not an infrequent complication. In one clinic of 265 cases of gunshot wounds of the head there were 21 cases, almost 10 per cent. The development of abscess is, of course, intimately related to the retention within the brain substance of infective material. Of all cases in which a bacteriological examination was made of a foreign body after extraction, some pyogenic organism was found in the culture media. (Cushing.) Consequently the argument in favor of the early removal of foreign bodies, together with the painstaking clearance of the tract, already referred to in relation to the incidence of meningitis and encephalitis, applies with equal force to that of abscess.

The acute abscess is the exception rather than the rule. The rapid disintegration of an infected area may eventuate into an abscess not long after the infliction of the wound, but in most instances abscesses of military practice are slow in their formation and result usually from retained foreign bodies. The interval from the date of

injury is often four or five months and in some instances longer. After the latent period, so characteristic of brain abscess, the typical symptoms indicating brain pressure develop insidiously. Forewarnings may have been given by an occasional rise of temperature, signs of irritability, or other temperamental changes, perhaps an increase of certain signs of focal disturbance, such as hemiplegia or aphasia. In the localization of the abscess the detection of a foreign body by the X-ray may serve as a valuable guide. The exploration should be made with a sharp bistoury and the abscess cavity, if found, drained with rubber tubing. The employment of a perforated metal tube has been recommended by English surgeons. This allows the pus to escape freely through the perforations into the tube and the latter prevents the collapse of the drainage tract. At each daily dressing the contents of tube are evacuated, and the tube filled with glycerin.

Because of the possible damage from pressure upon the surrounding brain tissue, tubular drainage should be dispensed with as soon as possible, substituting gauze wicks saturated with glycerin. Recurrence of symptoms such as fever, headache, vomiting, or focal signs presupposes inadequate drainage or loculation. Under such circumstances the abscess cavity may be explored gently with the finger to search for pockets or recesses. The obliteration of the cavity by granulation tissue is essentially a slow process, and weeks or months may elapse before this has been accomplished. The mortality is high in military as in civil practice. About 50 per cent of the cases terminate fatally. The rupture of the abscess into a ventricle is always disastrous.

Epilepsy.—The incidence of epilepsy as a sequel of gunshot wounds of the head has not been so great as was anticipated. With the lapse of time, however, a larger percentage of cases will be reported. There is nothing to be gained from speculating as to the pathogenesis. Suffice it to say that the occurrence of epilepsy seems to bear no relation to the site, depth, extent, or severity of the initial injury. It has been noticed, however, that the disease develops more frequently in cases in which there has been a long-standing wound infection. The administration of bromides for months to patients who have sustained head injuries has been advocated as a really effective prophylactic measure. Once the epileptic seizures have developed, the treatment will depend upon the nature of the lesion. The problems now in no wise differ from those to which the surgeon in civil practice is accustomed. This exception may be made, however, that a large number of epileptics from the war zone have cranial defects and these lesions offer a more frequent indication and opportunity for surgical intervention.

Cranioplasty.—The train of symptoms of which patients with cranial defects complain is endless. Some of them, no doubt, may

have a physical basis, but many are properly classified as traumatic neurosis. Among the more common symptoms are headaches, vertigo, sudden blurring of vision, especially on stooping, objection to loud noises, inability to sleep unless a firm bandage is applied to make pressure over the region of the defect. Usually at the site of the injury there is a sensitive scar, and at the center of the opening may be seen the transmitted pulsation of the brain, to describe which the term " pulsating skull defect " has been used.

The relation of the cranial defect to the etiology of epilepsy has been discussed chiefly in connection with the indications for cranioplasty. The bony defect itself can have no direct causative relationship, but the adhesions between the cicatrix, the meninges, and the cortex, may be an exciting factor in provoking epileptic seizures.

The indications for the performance of a cranioplasty are not very sharp and clear-cut and must be left to the discretion of the surgeon. When, however, the defect is large, when it involves the frontal bones with consequent disfigurement, when there is marked pulsation and bulging on coughing, when the cicatrix is tender and sensitive, and when the presence of the defect is an obsession to the patient, an operation for the repair of the defect may be considered justifiable.

The operation should not be performed until one year has elapsed since the injury, or at least six months and preferably a year after all signs of infection have abated.

In selecting material for the repair of a cranial defect, one must choose between foreign material, such as ivory or celluloid plates, and cartilage or bone. Obviously, preference should be given to cartilage or bone and the autograft is to be preferred to the heterograft.

The cartilaginous graft has some advantages over the bone graft. It is readily obtained from the cartilaginous ribs, is easily fashioned to any shape, is highly resistant to infection, does not shrink, is not absorbed, and usually maintains its vitality. Enough material may be removed from the seventh, eighth, and ninth ribs, and only half the thickness of the rib is required. To prevent the grafts from slipping out of the defect a trellis of fine gut is prepared by running a catgut suture across and back in a zigzag manner from the pericranium on one side to that on the other until the space is covered. To prevent adhesions the perichondrial surface should be faced toward the dura.

Bone grafts have been secured from the scapula, tibia, and skull, preferably from the latter in the parieto-occipital region. The steps of the operation are as follows: The region is exposed by a horseshoe flap. The shape and size of the graft is outlined by an incision in the pericranium, using a pattern cut the exact shape and size of the defect to be repaired. With a broad, flat chisel a thin shell of the outer table, together with the pericranium, is chiseled

off, working always from the periphery toward the center. When free, the graft is transferred to the defect, previously prepared for its reception, and secured in place, pericranium downward, with six or eight interrupted catgut sutures through the pericranium.

The exposure of the defect is made preferably with a curved flap incision at least a centimeter wide of the margins. The flap exposure is much to be preferred to the crucial incision.

The preparation of the defect for the reception of the graft is a matter of considerable importance. The adherent dura and scar tissue must be freed from the bony margins and entirely removed from the defect if this can be accomplished without damage to the cortex. Often a cortical subarachnoid cyst will be found beneath the dura and may be evacuated. The sclerotic bony margins are removed with a chisel so as to leave a leveled surface.

Before the graft is inlaid, bleeding must be controlled from every source. This is of the utmost importance. The graft, whether bone or cartilage, is then transferred in the manner above described.

If the operation is performed under every aseptic precaution the results will be uniformly good. In from six to eight weeks the process of bone regeneration will have advanced so far that consolidation of the defect will be complete.

CHAPTER VI.

BRAIN TUMORS.

Hospital records show that the brain is as frequently the seat of tumors as are other organs. The appended tables published by H. Tooth (*Brain*, 1912, xxxv, 61–108) contain in small space much data which is valuable.

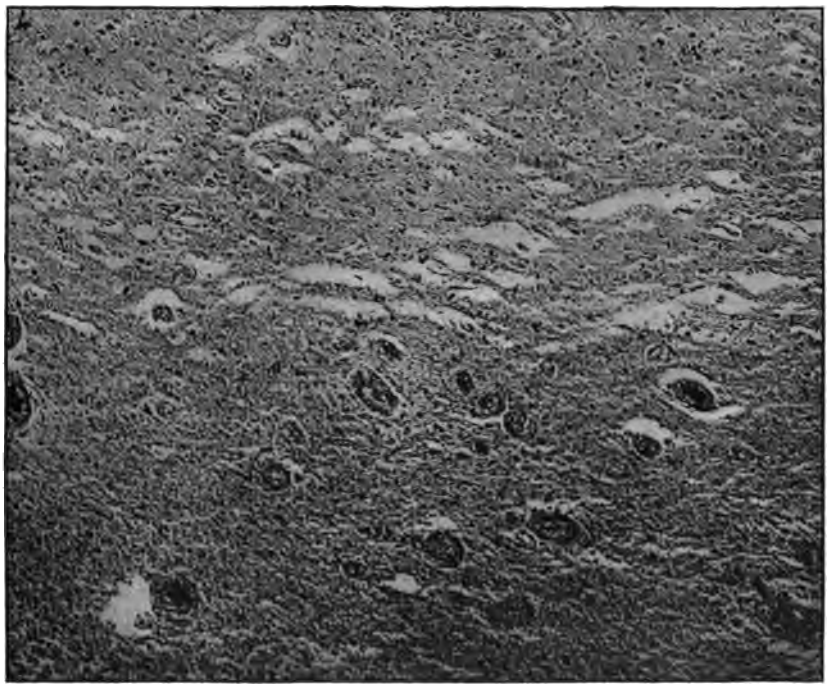

FIG. 74.—Illustrating gradual infiltration of glioma into adjacent brain tissue. Note edema of non-infiltrated tissue. × 80. (Bagley.)

Glioma.—This is the most common of brain tumors. In Tooth's series of 258 cases, 49.2 per cent were gliomata. These are benign growths, in the sense that they do not give rise to distinct metastases, but they are extremely destructive, often infiltrating most important nervous tissue. The tumors vary in size from very small nodules to huge masses which occupy the greater part of one hemisphere of the cerebrum. They are rarely sharply defined. Extensive infiltration of the nervous tissue is the rule (Fig. 74), but in the peripheral

parts of some gliomata there is a tendency to the formation of fibrous stroma even to the extent of rough capsulation. Gliomata develop from neuroglia and are found in any portion of the brain, spinal cord, and retina.

Histologically, these tumors consist chiefly of cells resembling neuroglial elements, small cells with numerous delicate branching processes, but with these there is usually a number of spheroidal cells

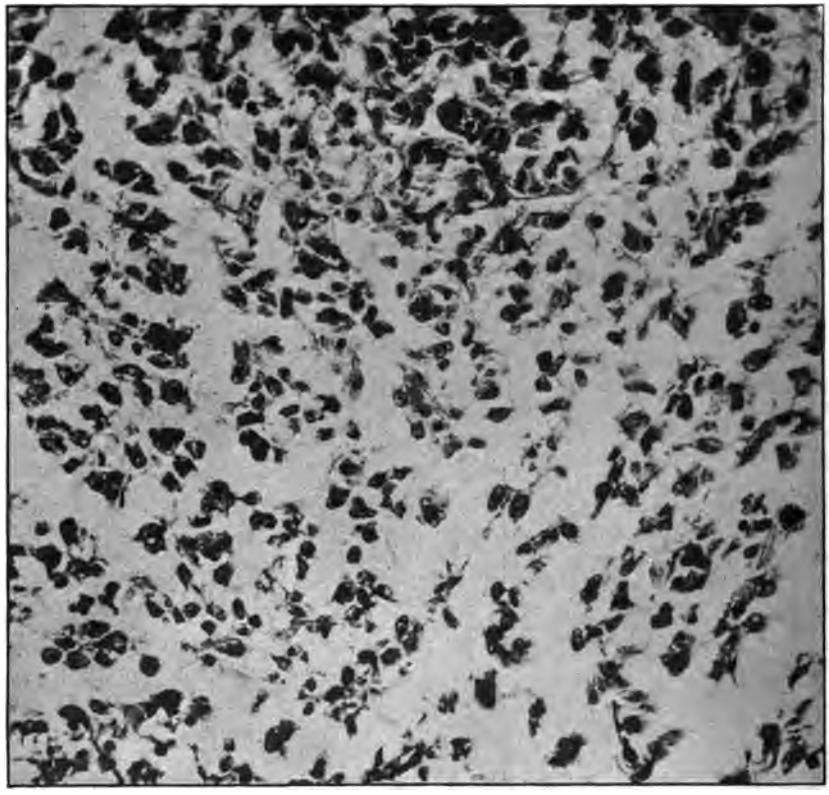

FIG. 75.—Illustrating cellular arrangement of glioma. × 500. (Bagley.)

with proportionately large nuclei. The question of the independence of some of the delicate fibrils at the edge of a cell or lying free is still unsettled.

Gliomata are classed with slow-growing tumors. Striking exceptions to this rule have been observed; for example, patients not infrequently exhibit a marked degree of increased intracranial pressure four weeks after the initial symptoms, and in one such case death occurred two months after the first symptom. The autopsy in this case revealed a very extensive involvement of the right cerebral hemisphere.

The tumors are prone to cystic and fatty degeneration, necroses, and hemorrhagic extravasations. Gliomatous cysts vary from

FIG. 76.—Illustrating interstitial hemorrhage in diffuse glioma. × 500. (Bagley.)

small microscopic spaces to large cavities containing 50 to 60 c. c. of fluid. The cyst walls are well defined, usually without a

FIG. 77.—Frontal view of endothelioma. Concentric appearance at apex the seat of outgrowth from dura. (Bagley.)

definite cellular lining. The cyst content is a straw-yellow fluid with high albumin and globulin percentage; on standing, a definite

coagulum forms. The cystic process may progress to a high degree and in some instances, in the slow-growing tumors, entirely replace the original neoplasm. Clinically, the change is an important factor, since the cysts are easily evacuated and complete extirpation often not difficult. Extensive cyst formation is seen more frequently in tumors of the cerebellum than of the cerebrum. Fatty degeneration of gliomata is seen too infrequently and in too limited

FIG. 78.—Fronto-lateral view of specimen in fig. 77. Note line of demarcation above and below and large cavity posterior. The cavity was filled with blood clot which resulted from the rupture of a vessel of the pia crowded backward during the invagination of the tumor. (Bagley.)

extent to justify attaching great importance to the change. Necrosis, commonly encountered in tumors of large size, is the result of pressure, thrombosis, etc. The brain tissue surrounding gliomata may also through much pressure be the seat of necrosis.

Owing to the abundance of thin-walled blood vessels and the softness of the growth, they are liable to interstitial hemorrhages. These vary from microscopic extravasation to collections sufficiently large to cause death through sudden increase of intracranial pressure.

Endothelioma, less common than glioma, is usually firm, surrounded by a firm capsule, and does not infiltrate the surrounding nervous tissue. These tumors, in most instances, originate in the

FIG. 79.—Sagittal section through tumor and frontal lobe of fig. 77 illustrating invagination. (Bagley.)

endothelial cells of the blood vessels and dural sinuses. Endothelial cells are normally elongated and thin, forming a mosaic over the surface which they line. In endotheliomata the cells seldom resemble

normal endothelium, but are cuboidal, polyhedral, or even cylindrical in shape, or on pressure may assume elongated, fusiform, or other bizarre forms. The varied arrangement of the cellular elements of tumors classed as endothelioma has resulted in much confusion and has made impossible a sharp line of classification. Cushing (Tumors of the Nervus Acusticus, etc.), in discussing the histology of acoustic tumors, the majority of which he includes in the endothelial group, gives in some detail the result of careful study of a number of tumors. Cushing and his co-workers concluded that the tumors are composed of two types of tissue, the one of dense interlacing fibrous

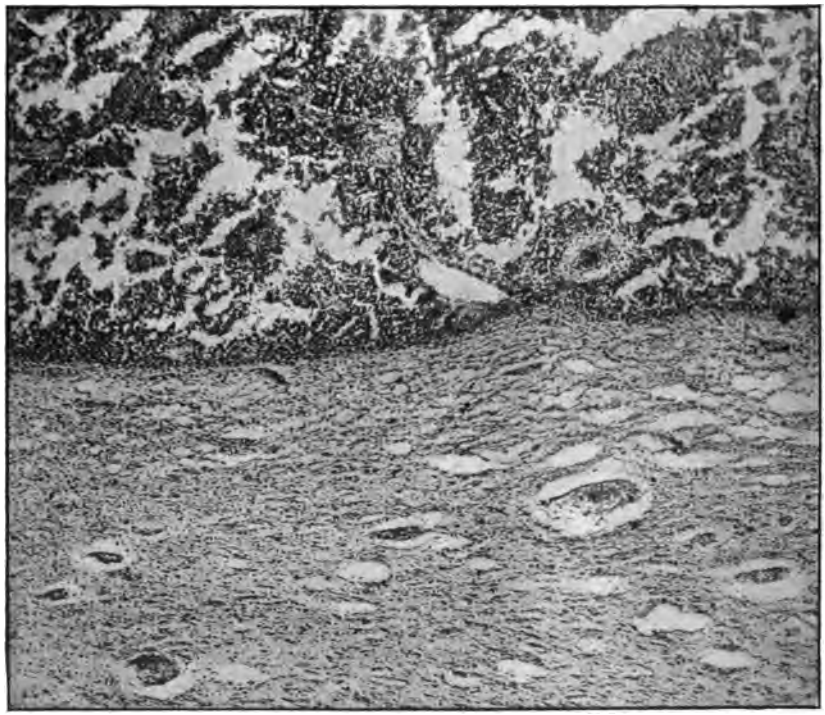

FIG. 80.—Illustrating sharp line of demarcation of endothelium and adjacent brain. Section removed from b. in fig. 77. (Bagley.)

bands, the other of loose reticular tissue. The fibrous bands are composed of masses of elongated cells with oval nuclei. The reticular tissue, on the other hand, appears in areas of greater or less extent between these fibrous bands and has an edematous or hydropic appearance with relatively scant round cells of varying size, with sparse protoplasm lying in a loose tissue network.

Endotheliomata are slow-growing tumors. They may occur in almost any portion of the brain but are frequently found in the cerebellopontile angle. These tumors are often the seat of degenerative

processes. The most frequent are gelatinous cysts or calcified bodies of various sizes. When the latter occupy the greater portion of the neoplasm, the tumor is spoken of as psammoma. Because of their firm encapsulation, the frequency of extracerebral or extracerebellar origin, the fact that the tumors merely crowd nervous tissue, these of all tumors are most favorable for operative removal.

Sarcoma is a tumor arising from connective tissue. Therefore, those classified with brain tumors may be of intra- or extra-cerebral origin. They are firm, entirely without or very poorly provided with a capsule, and are easily distinguished from the brain tissue through

FIG. 81.—Illustrating fibrous-like structure of endothelioma. *a*. point of development from pia as shown in fig. 77. (Bagley.)

their increased vascularity. Histologically their structure depends upon the tissue from which the particular growth springs, but it must be remembered that it is scarcely possible, from the study of the microscopic preparation, to determine the type of mother cells. The stroma of these tumors may predominate and give rise to a very firm tumor simulating fibroma, or they may be very rich in cells and result in soft pulpy masses. There are several mixed varieties, such as osteo-sarcomata, myo-sarcomata, psammo-sarcomata, and melano-sarcomata.

Diffuse sarcoma, involving the meninges of the entire nervous system, is occasionally encountered. The lesion may be due to rupture in the ventricle of a sarcomatous mass.

The brain and spinal cord may be involved in secondary sarcomata. These tumors are difficult to remove, and the post-operative results are very disappointing.

Tuberculoma.—Tuberculoma of the brain is usually in the form of circumscribed masses of tissue varying from very small masses to accumulations several centimeters in diameter. The lesion is frequently secondary to tuberculosis elsewhere in the body. As may be expected, the lesions are frequently multiple. They are slow growing and, depending upon their location in the brain, may exist a number of months before producing symptoms. Tuberculomata generally consist of a dense central cheesy mass, around which is a zone containing tubercular granulation, numerous small spheroidal cells and giant cells. Operative removal of solitary tubercles is frequently a simple procedure, but the results are disappointing, since there is in all probability one or more similar lesions at varying stages of development. Tuberculoma may be the seat of extensive necrosis.

Gumma.—Gummata, though suspected in almost every case presenting tumor syndrome, are actually seldom encountered clinically or at autopsy. The writer observed at operation a well localized caseous mass in the region of the operculum in a patient who presented marked speech disturbance and symptoms of increased intracranial pressure. The tumor was uncovered by Dr. Cushing, the caseous mass removed without destruction to the surrounding cerebral tissue. The operative recovery was most satisfactory. Complete medical treatment was instituted during convalescence, with complete recovery. The symptoms in this case were exactly similar to what would have been expected had any other cerebral neoplasm originated in the location.

Carcinoma.—Carcinoma of the brain and spinal cord is found secondary to carcinoma elsewhere in the body, frequently following primary lesions in the stomach, lungs, and breast. (Fig. 82.) The lesions may occur in the skull or any portion of the brain or its coverings, and are frequently multiple. Removal should not be attempted, for obvious reasons.

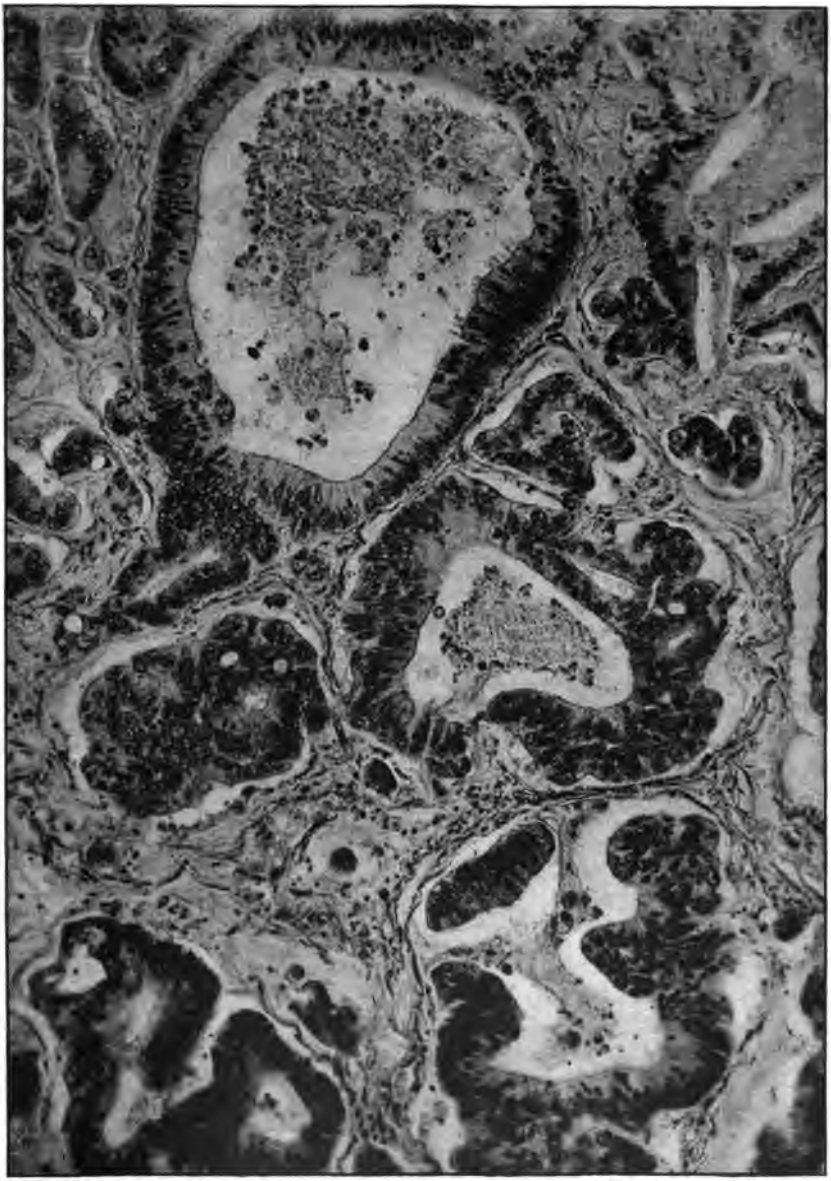

FIG. 82.—A section of a brain tumor which was secondary to an intestinal carcinoma. × 175. (Bagley.)

TABLE III.—*Age at appearance of first symptoms for growths of which the nature has been verified on operation or post-mortem.*

Nature of growth.	1 to 20 years.				21 to 30 years.				31 to 40 years.				41 to 50 years.				51 to 60 years.				61 to 70 years.				Total.	Per cent.
	Male.	Female.	Total.	Per cent.	Male.	Female.	Total.	Per cent.	Male.	Female.	Total.	Per cent.	Male.	Female.	Total.	Per cent.	Male.	Female.	Total.	Per cent.	Male.	Female.	Total.	Per cent.		
Glioma	9	16	25	19.6	25	12	37	29.1	25	7	32	25.1	14	5	19	14.9	8	5	13	10.2	....	1	1	0.7	127	49.2
Fibroglioma	....	2	2	13.3	....	4	4	26.6	2	4	6	40.0	1	2	3	20.0	....	....	....	.....	....	....	....	.....	15	5.8
Fibroma	2	2	4	30.7	....	....	....	.....	7	2	9	69.2	....	....	....	.....	....	....	....	.....	....	....	....	.....	13	5.0
Endothelioma	4	2	6	16.2	2	2	4	10.8	8	8	16	43.2	7	2	9	24.3	1	1	2	5.3	....	....	....	.....	37	14.3
Sarcoma	3	....	3	14.2	5	....	5	23.8	5	1	6	28.5	3	3	6	28.5	....	1	1	4.7	....	....	....	.....	21	8.1
Carcinoma	....	....	....	.....	....	....	....	.....	3	....	3	20.0	3	3	6	40.0	3	2	5	33.3	1	....	1	6.6	15	5.8
Tuberculoma	4	5	9	64.2	2	2	4	28.5	1	....	1	7.1	....	....	....	.....	....	....	....	.....	....	....	....	.....	14	5.4
Cyst, simple	3	....	3	60.0	2	....	2	40.0	....	....	....	.....	....	....	....	.....	....	....	....	.....	....	....	....	.....	5	1.9
Papilloma	....	....	....	.....	1	....	1	33.3	....	....	....	.....	1	1	2	66.6	....	....	....	.....	....	....	....	.....	3	1.1
Cholesteatoma	....	....	....	.....	....	1	1	.....	1	....	1	.....	....	....	....	.....	....	....	....	.....	....	....	....	.....	2	.7
Pituitary	....	....	....	.....	1	....	1	.....	1	....	1	.....	....	....	....	.....	....	....	....	.....	....	....	....	.....	2	.7
Pineal	1	....	1	.....	2	....	2	.....	....	....	....	.....	1	....	1	.....	....	....	....	.....	....	....	....	.....	4	1.5
Total	26	27	53	20.5	40	21	61	23.6	53	22	75	29.0	30	16	46	17.8	12	9	21	8.1	1	1	2	.7	258	.....

TABLE IV.—*Regional incidence of growths verified on operation or post-mortem.*

Region.	Number of growths for each region.	Glioma.		Fibroglioma.		Fibroma.		Endothelioma.		Sarcoma.		Carcinoma.		Tuberculoma.		Cyst.		Papilloma.		Cholesteatoma.		Pituitary.		Pineal adenoma.	
		Number.	Per cent.	Number.	Per cent.	Number.	Per cent.	Number.	Per cent.	Number.	Per cent.	Number.	Per cent.	Number.	Per cent.	Number.	Per cent.	Number.	Per cent.	Number.	Per cent.	Number.	Per cent.	Number.	Per cent.
(1) Frontal	68	38	55.8	....	.....	....	.....	21	30.8	4	5.8	4	5.8	1	1.4	....	.....	....	.....	....	.....	....	.....	....	.....
(2) Central	43	25	58.1	....	.....	....	.....	10	23.2	3	6.9	5	11.6	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....
(3) Temporosphenoidal	27	17	62.9	....	.....	....	.....	4	14.8	2	7.4	2	7.4	2	7.4	....	.....	....	.....	....	.....	....	.....	....	.....
(4) Occipital	6	4	66.6	....	.....	....	.....	....	.....	1	16.6	1	16.6	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....
(5) Corona radiata, corp. callos., etc.	8	6	75.0	....	.....	....	.....	....	.....	1	26.5	1	26.5	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....
(6) Lateral ventricle	3	1	33.3	....	.....	....	.....	....	.....	2	66.6	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....
(7) Pituitary	2	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	2	.....	....	.....
(8) Optic thalamus	3	1	33.3	....	.....	....	.....	....	.....	2	66.6	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....
(9) Mesencephalon	8	4	50.0	....	.....	....	.....	....	.....	1	12.5	....	.....	2	25.0	1	12.5	....	.....	....	.....	....	.....	....	.....
(10) Pineal	4	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	4	.....
(11) Choroid plexus, III and IV ventricles	4	....	.....	....	.....	....	.....	....	.....	1	25.0	....	.....	....	.....	....	.....	3	75.0	....	.....	....	.....	....	.....
(12) Cerebellum	34	18	52.9	....	.....	1	2.9	1	2.9	3	8.8	1	2.9	6	17.6	4	11.7	....	.....	....	.....	....	.....	....	.....
(13) Extra-cerebellar	32	5	16.3	13	40.6	11	34.3	1	3.1	....	.....	....	.....	....	.....	....	.....	....	.....	2	6.2	....	.....	....	.....
(14) Pons	12	7	38.3	1	8.3	1	8.3	....	.....	....	.....	....	.....	3	24.9	....	.....	....	.....	....	.....	....	.....	....	.....
(15) Medulla	1	....	.....	1	.....	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....
(16) Base crus, etc.	3	1	33.3	....	.....	....	.....	....	.....	1	33.3	1	33.3	....	.....	....	.....	....	.....	....	.....	....	.....	....	.....
Total	258	127	49.2	15	5.8	13	5.0	37	14.3	21	8.1	15	5.8	14	5.4	5	1.9	3	1.1	2	0.7	2	0.7	4	1.5

CHAPTER VII.

TECHNIC OF STANDARD CRANIAL OPERATIONS.

The sub-temporal operation.—The following technic for sub-temporal openings is given in detail. For the development and operative details of this operation as here given, the credit is due Dr. Harvey Cushing. In the production of these photographs an attempt has been made to approximate his methods. The shaving of the scalp is best done the day of operation. The sterilization of the skin field may be completed after the anesthetic is begun and the patient is in proper position on the table. Scrubbing with green soap and water, followed by alcohol and bichlorid, is a satisfactory preparation. After sterilization of the field, and while the head is still uncovered, the line of incision may be scratched with a scalpel. The incision is 7 or 8 cm. in length and extends from just in front of the attachment of the ear up and very slightly backward, as shown.

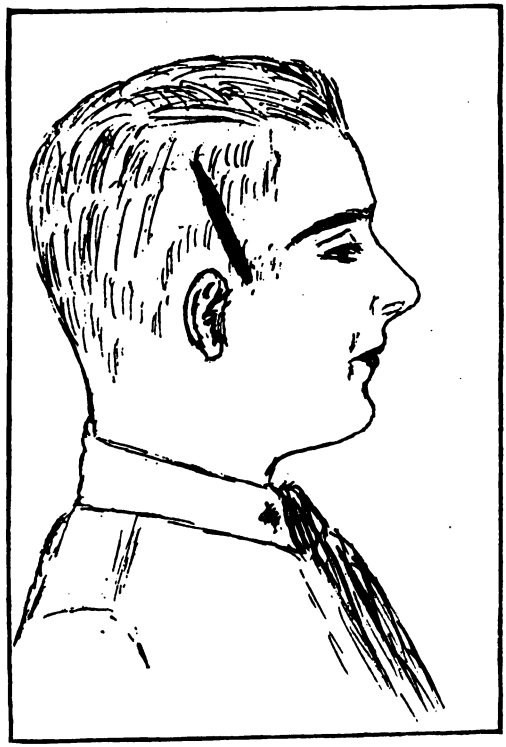

FIG. 83.

Then the entire head is covered by a double thickness of gauze saturated with bichlorid. The towels and sheets are arranged so that the minimum area of skin surface may be exposed and there will be no danger of disarrangement in the course of operation.

The scratch indicating the incision is exposed by cutting the gauze after the draping is completed.

The method here shown for controlling hemorrhage controls all but the smallest amount of bleeding if properly performed. The

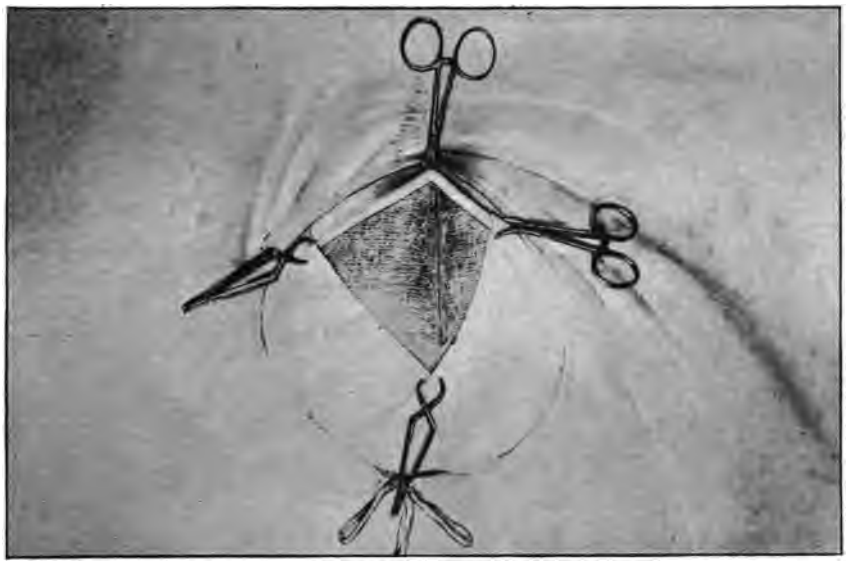

FIG. 84.

FIG. 85.

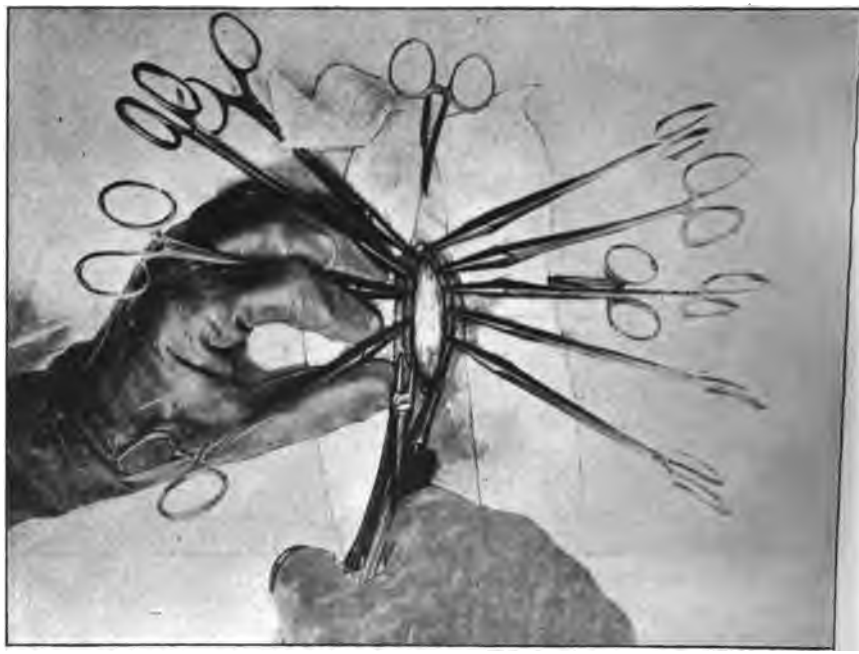

FIG. 86.

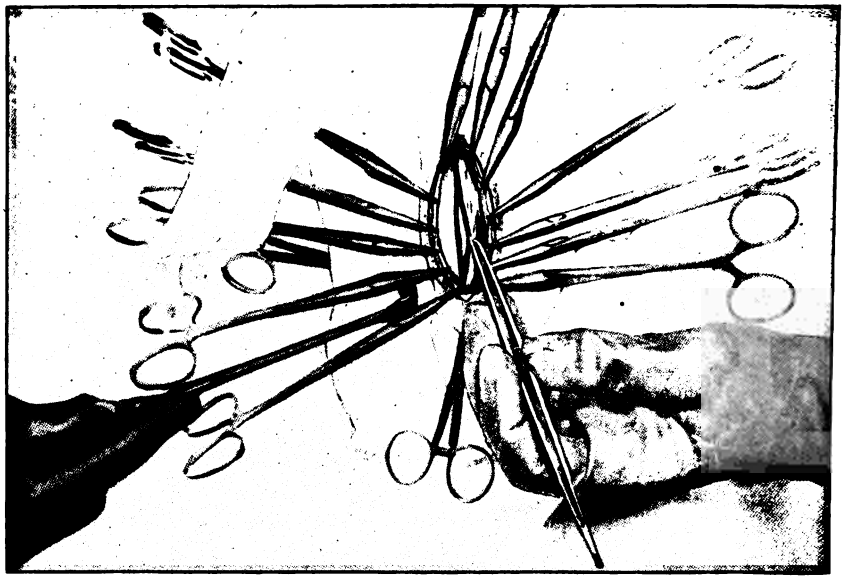

FIG. 87.

operator's left hand is shown controlling the hemorrhage from one side of the incision. Opposite it is the left hand of the assistant, while the index finger of the assistant's right hand exerts pressure over the temporal artery. By pressing the scalp against the underlying bone and pulling it, at the same time, away from the site of incision, hemorrhage can be controlled.

The incision is extended through the thickness of the scalp and the galea can be separated from the underlying temporal fascia. A

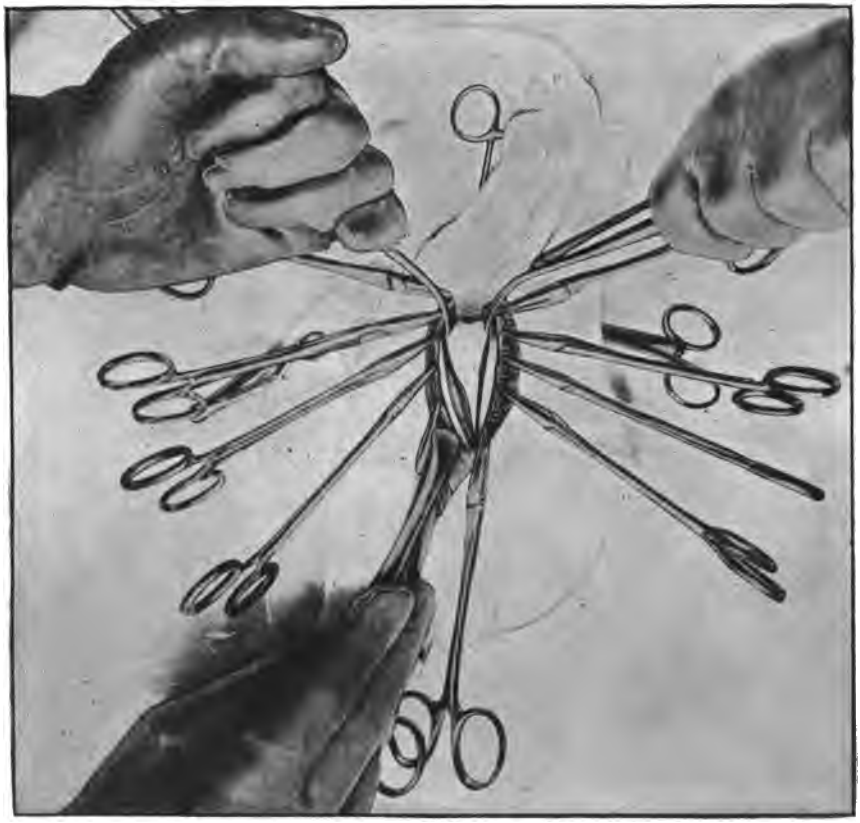

FIG. 88.

tourniquet is not needed to control bleeding in this type of operation, and digital pressure answers the purpose.

At the lower end of the incision the temporal artery or its larger branches are sometimes cut. Before the pressure of the finger is released, straight-pointed hemostats are clamped to the galea at intervals of about 1 or 1.5 cm. These hemostats, when reflected back over the scalp, will control all bleeding without the vessels themselves actually being caught.

Incision is next carried at one cut through temporal fascia and muscle to bone. The line of incision is in the same direction as that previously made in the scalp. Interlacing of fibers of temporal muscle prevents proper exposure from being obtained if an attempt is made to split the muscle without cutting. Care must be taken to free the attachments of the fascia from the bone at the upper end of the incision. Tissue forceps are shown picking up the temporal

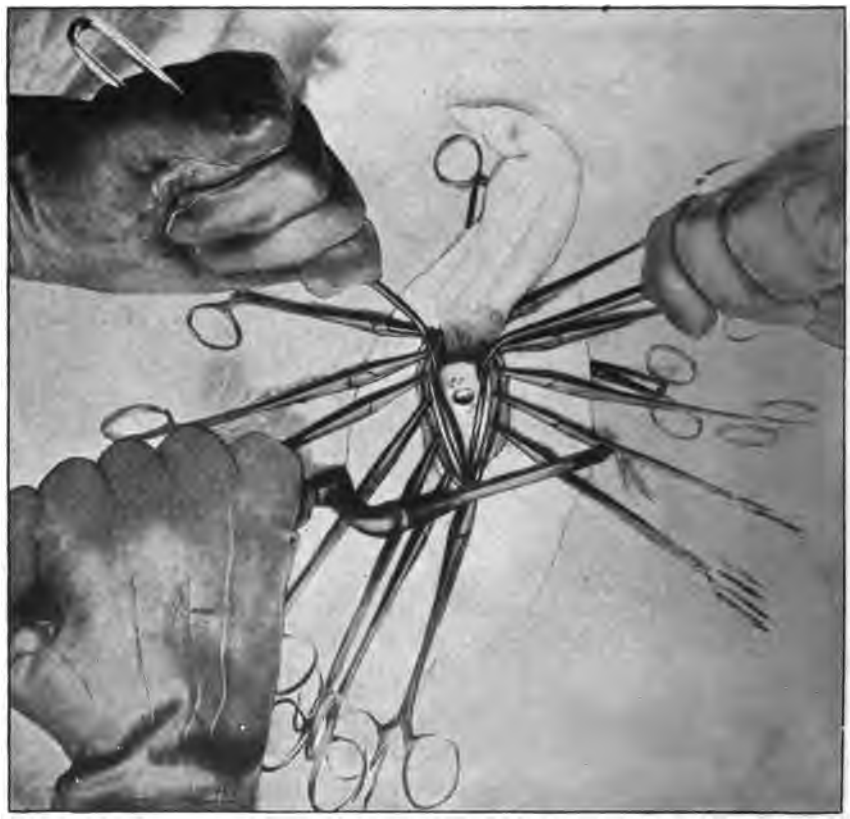

FIG. 89.

fascia after the incision has been made. The muscle has also been cut through and the bone is shown beneath. The small type of claw retractors can be used best in this stage of the operation.

With a square-pointed periosteal elevator the muscle is separated from the squamous portion of the temporal bone over an area of about 8 or 9 cm. in diameter. At the lower end of the exposure bleeding may be encountered which is readily controlled by tucking a gauze sponge between the temporal muscle and the bone.

The area of bone which has been uncovered can best be reached by the use of double-angled retractors as shown in figure 88. With these retractors the muscle and fascia can be lifted from the surface of the bone. A bone opening is made with a large perforator. The perforator should have a broad, obtuse-angled cutting edge, and penetration of the bone is stopped when the point reaches the dura.

With a large burr the opening is enlarged. Only through gross carelessness can either the dura or the underlying brain be injured.

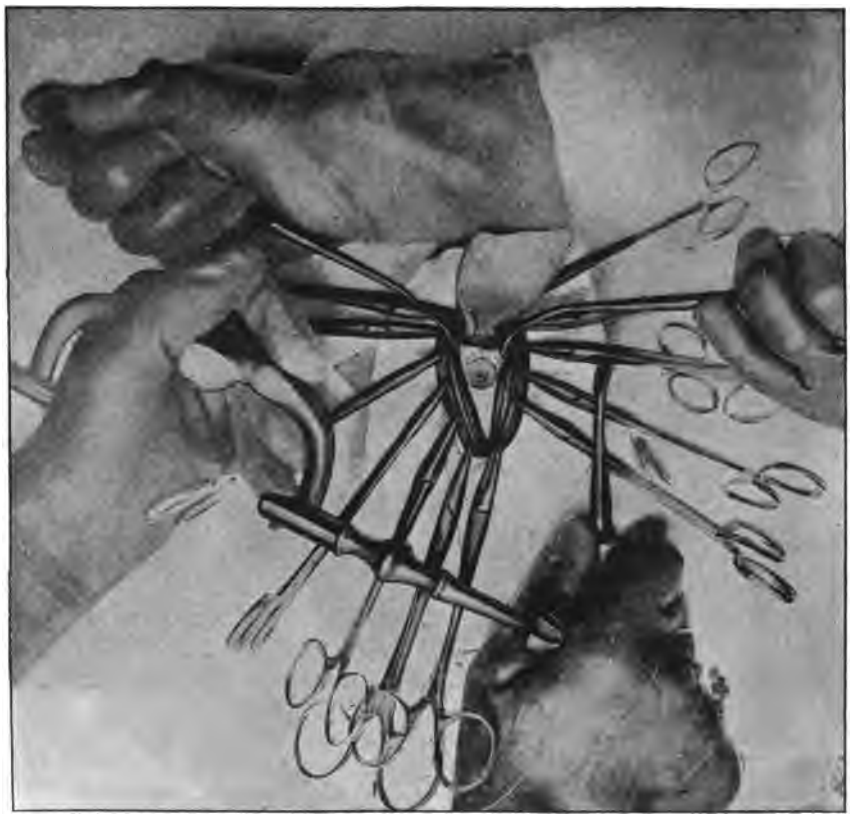

FIG. 90.

With blunt instruments the dura may be separated from the under surface of the bone, particularly downward and forward, where it should be done with care, as it is in this location that the middle meningeal vessels are found.

Not infrequently they groove the bone very deeply or, in special instances, may even tunnel through it. Enlargement of the bone opening in this direction should be preceded by separation of the middle meningeal vessels from the under surface of the bone. With small rongeurs the thin, squamous portion of the temporal bone is

removed. When the operation is conducted for drainage purposes, as in fracture of the skull with brain injury, a large opening is seldom necessary. When the sub-temporal operation, on the other hand, is performed to relieve a high degree of pressure, as is found in brain tumor, as large an opening as possible is made.

By the use of elevating retractors and small rongeurs a wide exposure of the dura can be obtained. In this stage of the operation care should be taken to break the bone outward and to exert no pressure on the dura. The use of cotton sponges is advised in all

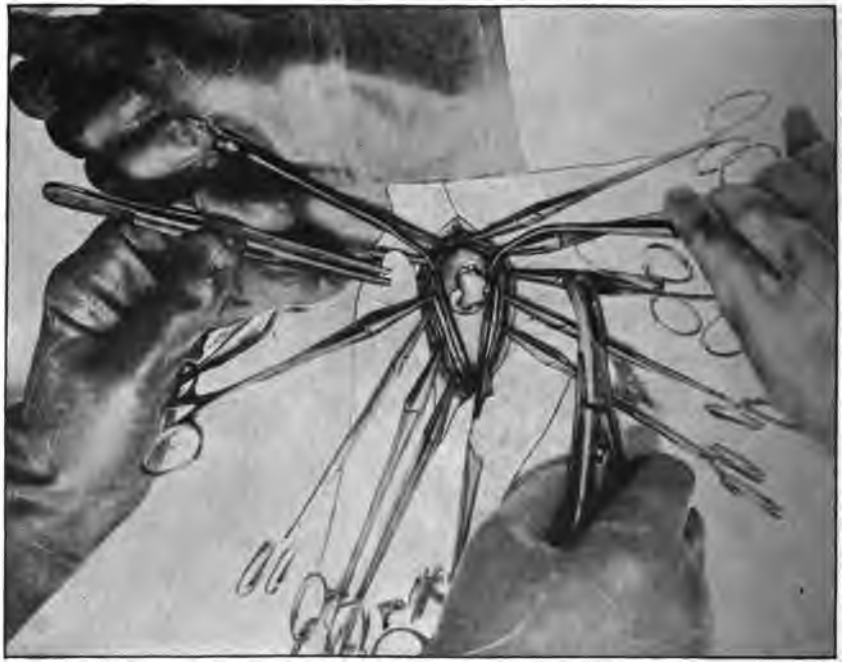

FIG. 91.

sponging, particularly if the brain surface is exposed. Through the somewhat translucent dura the larger cortical vessels can be seen.

In opening the dura a spot not overlying these vessels should be selected and the dura carefully cut with the belly of the scalpel until the pia-arachnoid is exposed. The opening can then be enlarged by the use of a grooved director.

When the opening is of sufficient size to introduce a brain spoon the dura may be cut radially with scissors back to the margin of the bone opening. Exploration can be conducted toward the base beneath the temporal lobe, clots removed from neighboring areas of the cortex.

At this stage any further operative procedure on the brain will be determined by the conditions for which the operation is performed.

Careful closure of the temporal muscle should be made with interrupted sutures of fine silk. Undue herniation of the brain is restrained by the temporal muscle and fascia, which also give protection and prevent obvious deformity.

The temporal fascia is then closed in the same way.

The closure of the galea is extremely important, and sutures should be placed at frequent intervals.

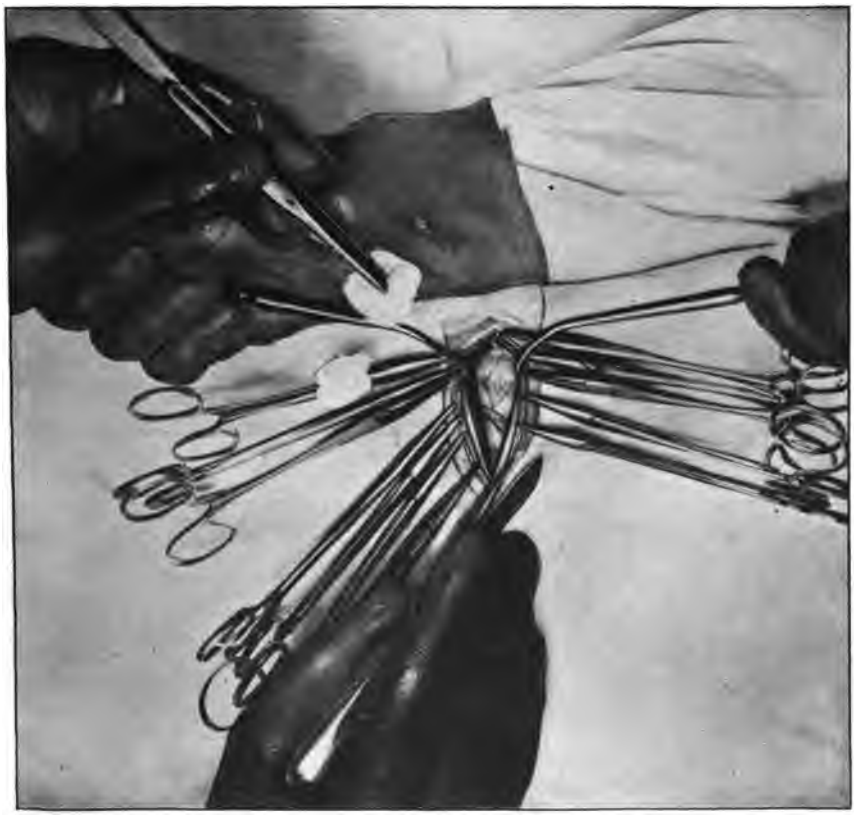

FIG. 92.

The final row of sutures may be placed with straight needles. The vessels of the scalp are controlled and bleeding is prevented by these last two rows of sutures which include all vessels between them.

Placement of the skin sutures on straight needles before any of them are tied, everts the skin margin and brings a broad area of the cut surface into approximation.

The final appearance of the wound is as shown. Note its elevated appearance. With the use of skin sutures of silk and with closure as given, the stitches should be removed at the end of 48 hours.

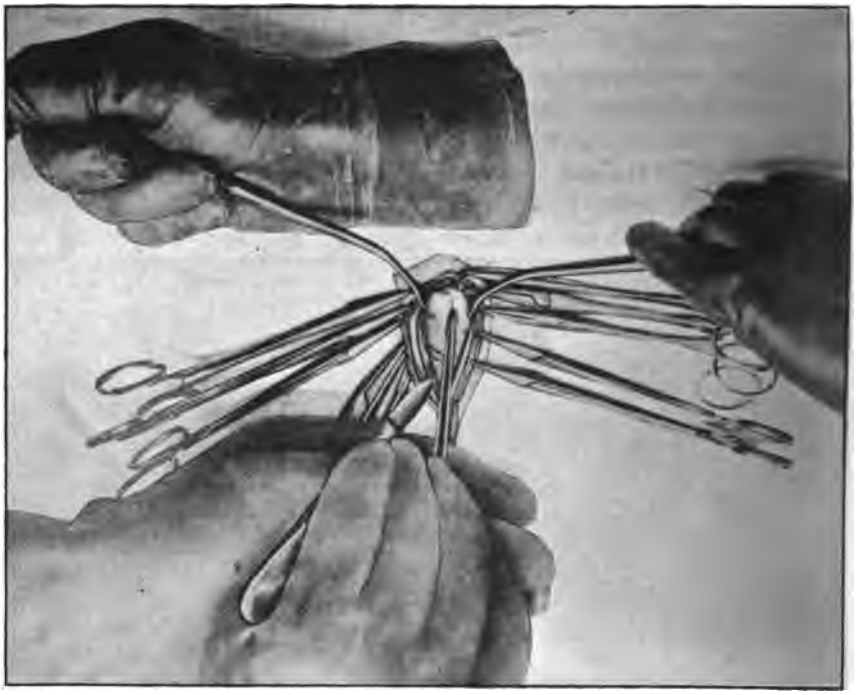

FIG. 93.

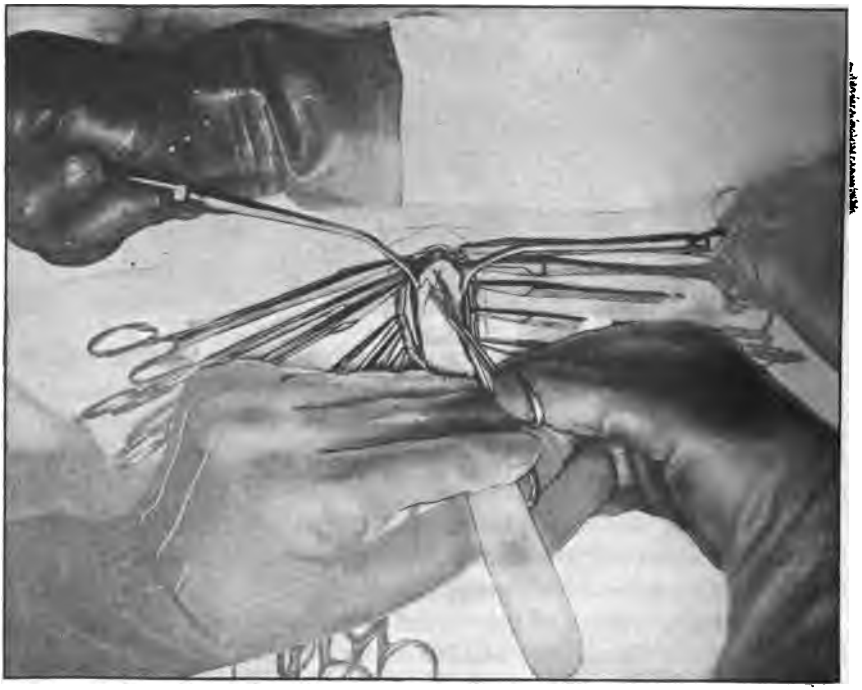

FIG. 94.

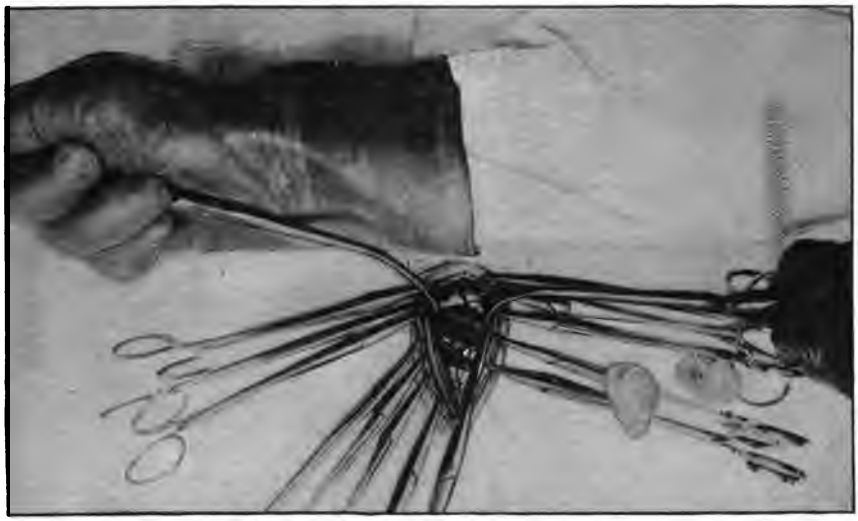

FIG. 95.

FIG. 96.

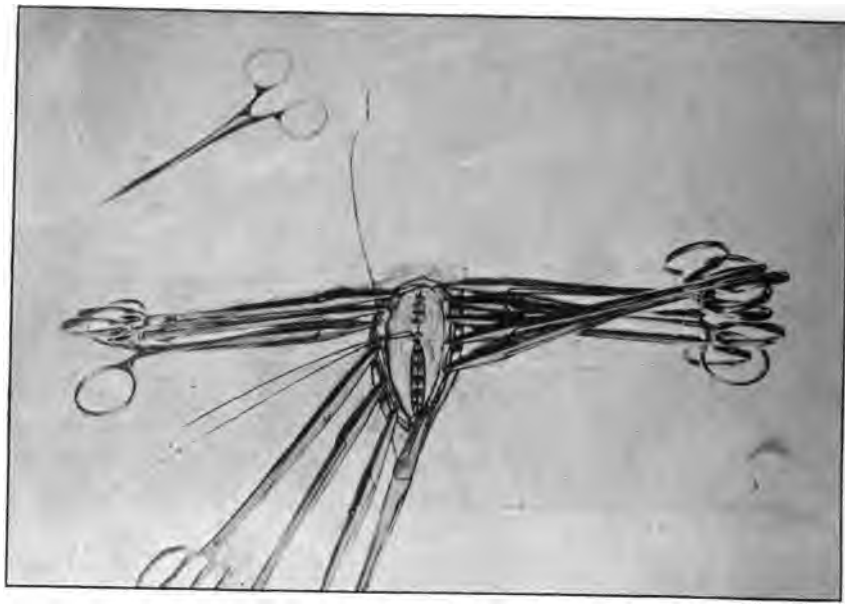

FIG. 97.

FIG. 98.

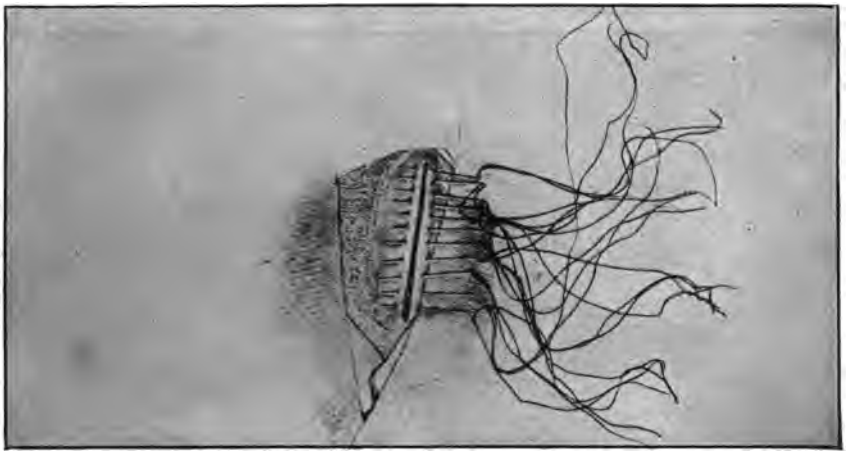

FIG. 99.

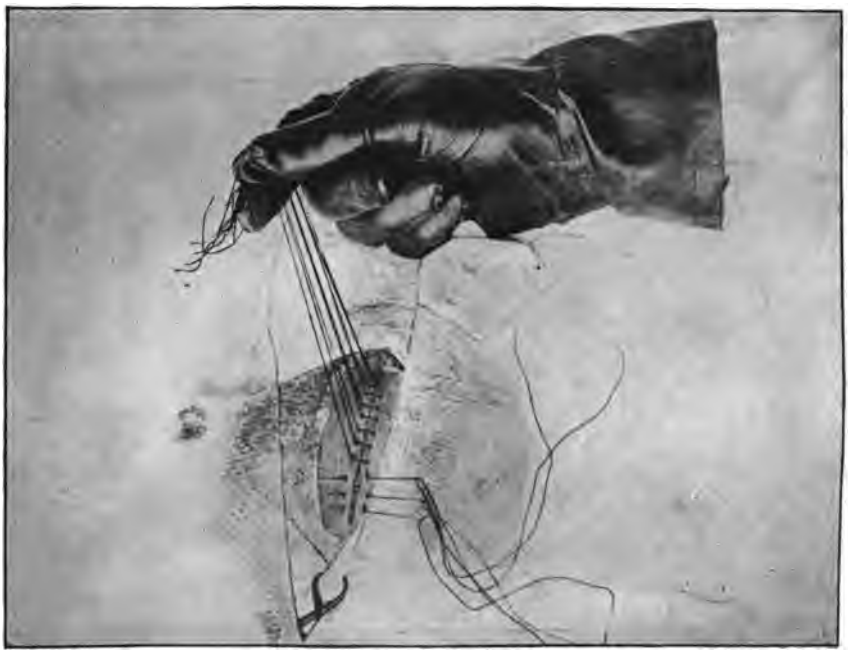

FIG. 100.

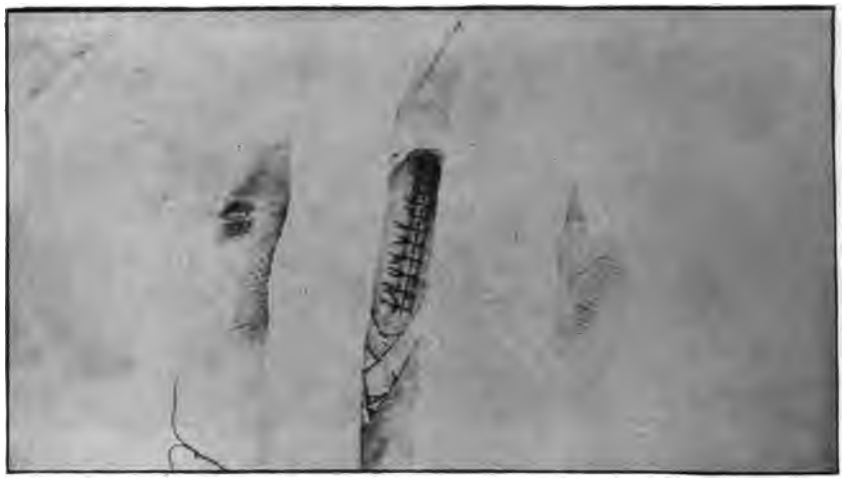

FIG. 101.

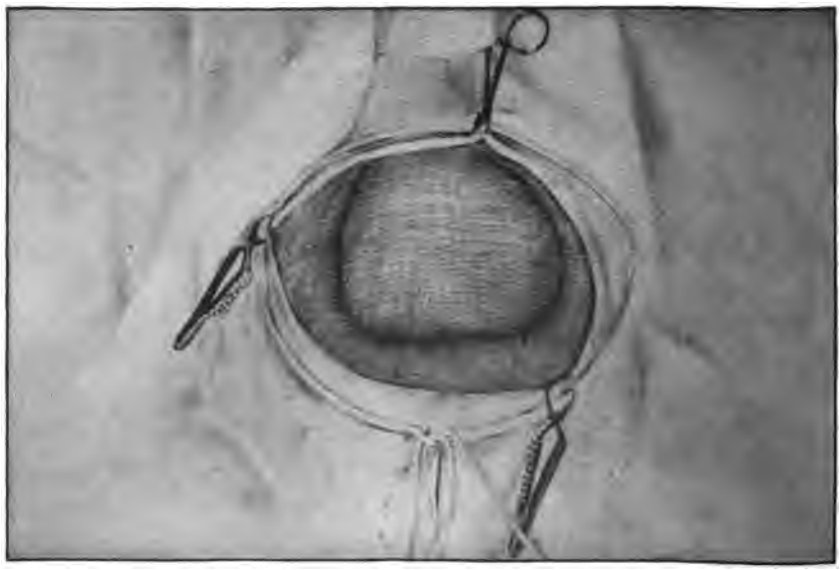

FIG. 102.

Osteoplastic craniotomy.—The following method of performing an osteoplastic craniotomy. is varied by the preferences of individual operators. Motor appliances may be used to facilitate certain steps, but the principles as here shown have come into general use. By some, tourniquets of various types are still used. They are not invariably satisfactory, and it is a frequent occurrence to see them removed in the course of an operation because of improper adjustment. Even if adjusted properly they may not control the oozing caused by the return flow of blood through the perforating vessels of the diploë. The method of control of hemorrhage from the convex or flap side of the incision as here presented has been found

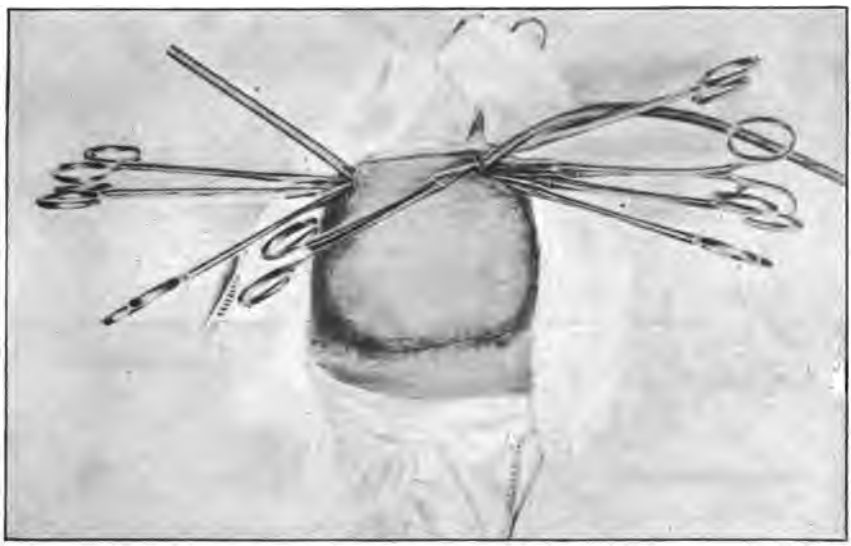

FIG. 103.

practicable and avoids the cumbersome number of hemostats otherwise used. The preparation of the field of operation is as indicated in the sub-temporal operation. The proposed incision is scratched on the skin.

The base of the flap is always made in the sub-temporal region, as at this place the squamous portion of the temporal bone is thin and has no diploë. Also the temporal muscle and fascia serves as a hinge. Small incisions are made at the end of both limbs of the proposed incision. These are carried through scalp, temporal fascia and muscle down to bone. A properly curved periosteal elevator is inserted through one of them along the bone and out through the other opening. A rubber catheter is then passed through.

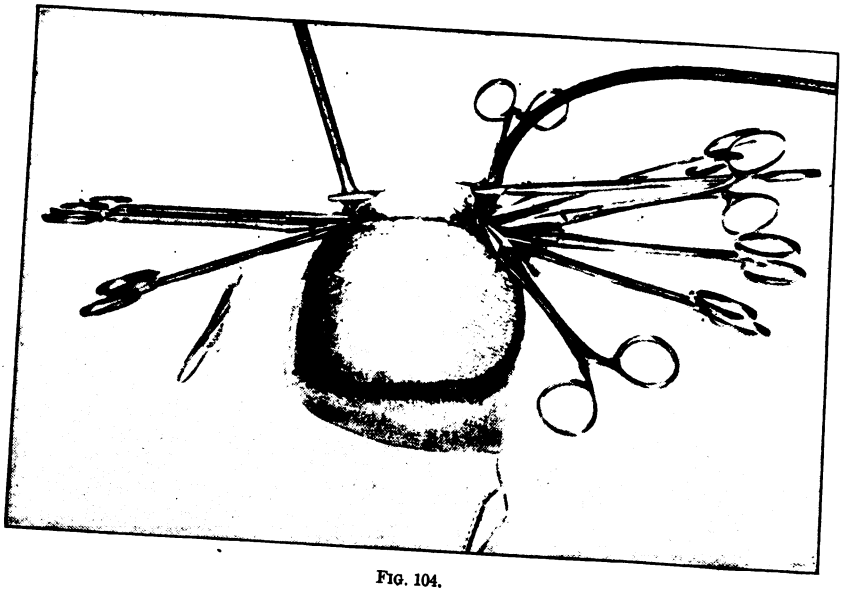

FIG. 104.

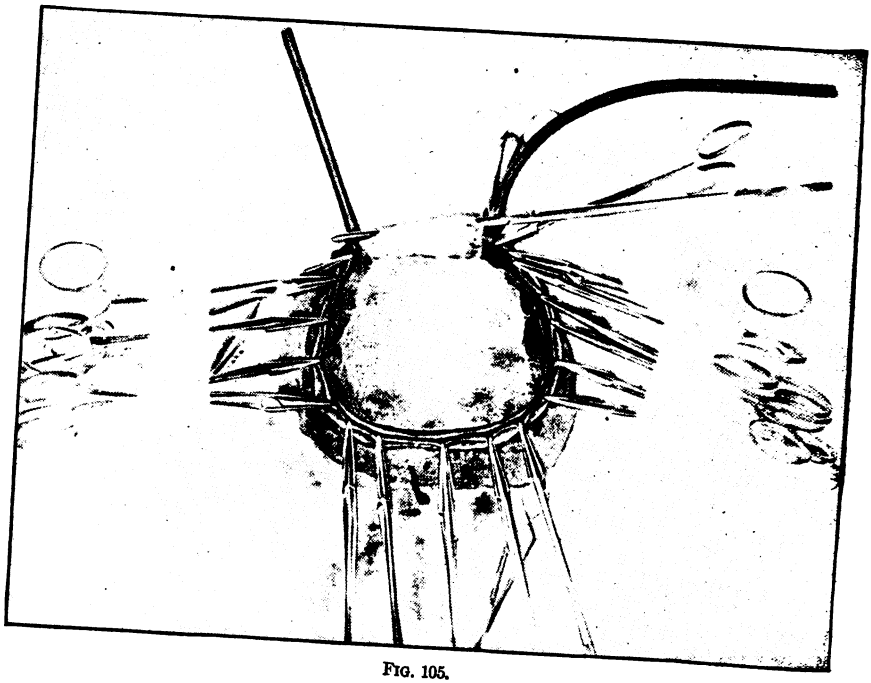

FIG. 105.

A small guaze sponge is placed over the base of the flap to prevent crumpling of it, the catheter is tightened and the tension maintained with a straight clamp as shown. By some such simple procedure the bleeding from the flap itself is permanently controlled. The degree of constriction exercised by the catheter can be made just sufficient to control the bleeding from the flap side.

The entire incision can then be completed, the hemorrhage from the convex side being controlled by digital pressure all hemo-

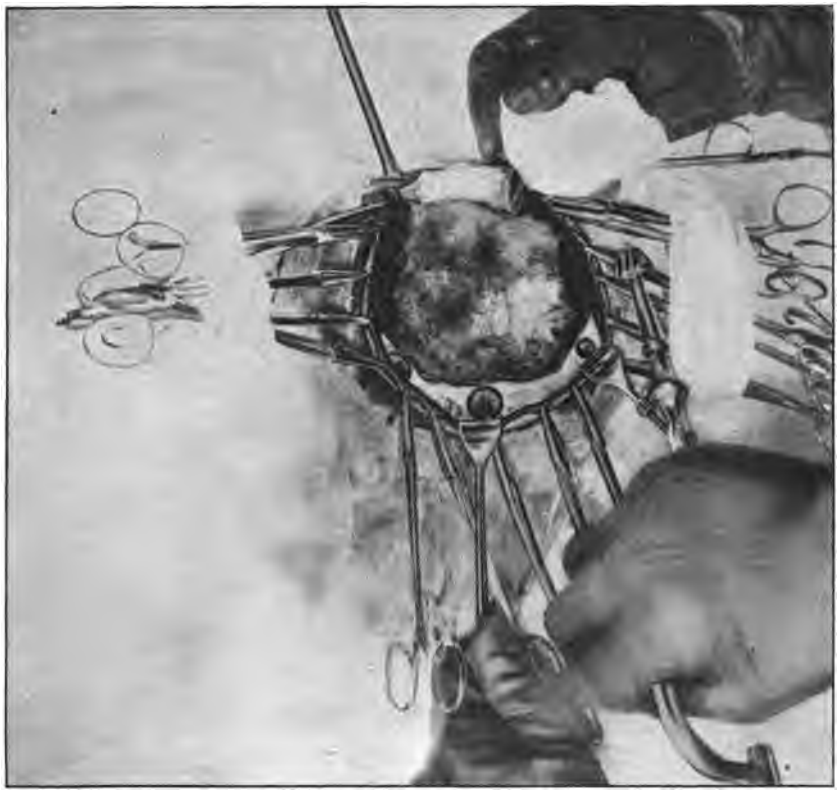

FIG. 106.

stats are placed on the galea and are reflected over the cut edge of the scalp.

Three primary openings are made with a perforator and enlarged by the use of a burr, as shown.

At this stage a blunt instrument may be used to separate the dura from the bone between these openings. Some oozing may follow this step, but the intra-cranial pressure is usually sufficient to press the dura against the bone and control it.

The limbs of the bone flap are then cut with some bone cutting forceps. Of these the De Vilbiss type of forceps is more generally employed. The bone cuts are carried well down into the thin portion of the squamous bone of the temporal. Bleeding from the bone is usually satisfactorily controlled by the use of bone wax.

A Gigli saw guide is then passed between the perforator openings. The dura is protected by the insertion of an elevator and the saw drawn through.

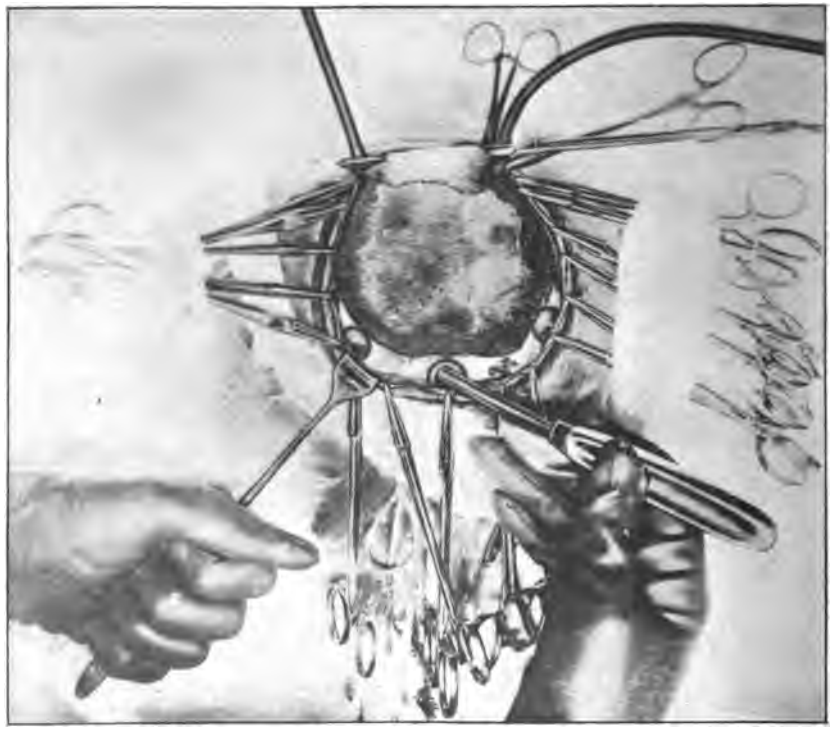

FIG. 107.

The bone is cut from within out, care being taken to make a broad beveled edge.

With blunt instruments the flap may then be raised and broken across its base. At this stage it may occasionally happen, particularly in those instances in which the middle meningeal arteries tunnel the bone, that injury to it occurs.

Prompt pressure or a small pack between the dura and bone will control this. The view of the catheter as it encircles the temporal muscle is also shown. At this stage the bone margin may be smoothed, or if it is desirable to secure decompressive results as well

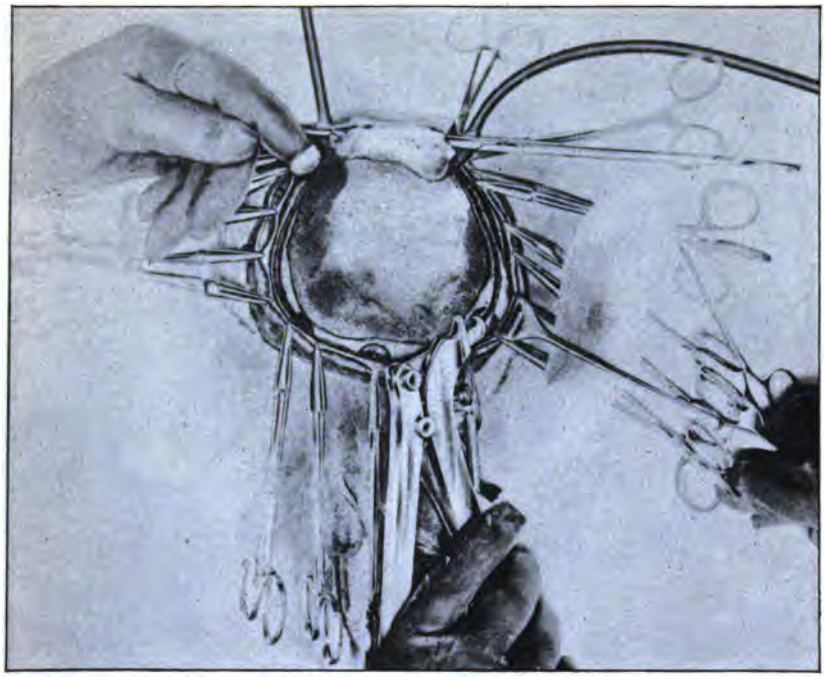

FIG. 108.

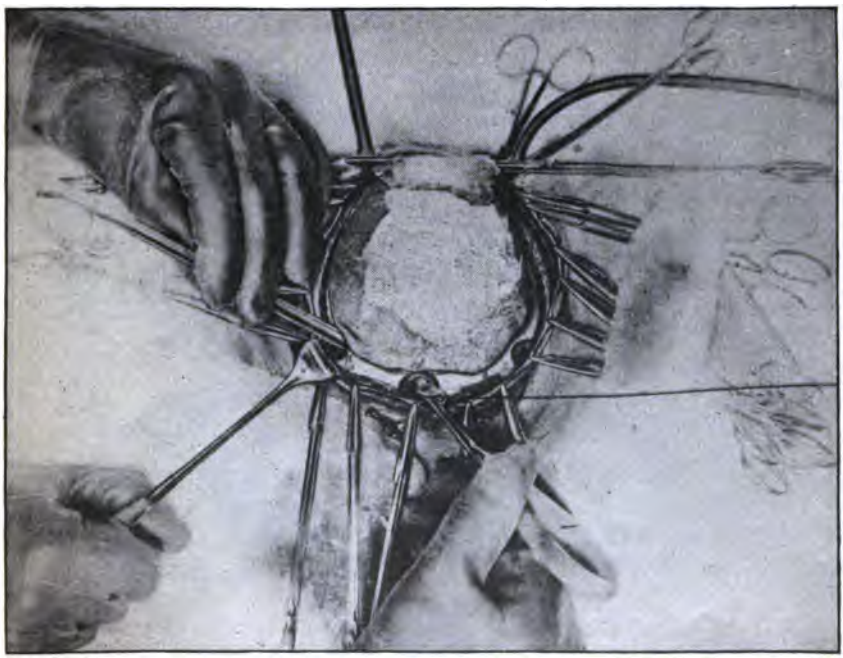

FIG. 109.

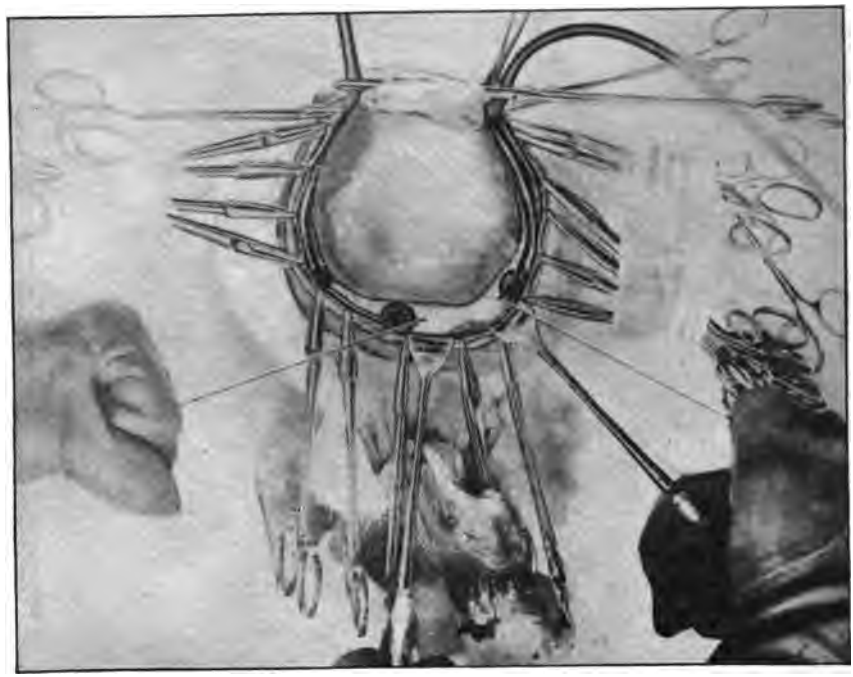

FIG. 110.

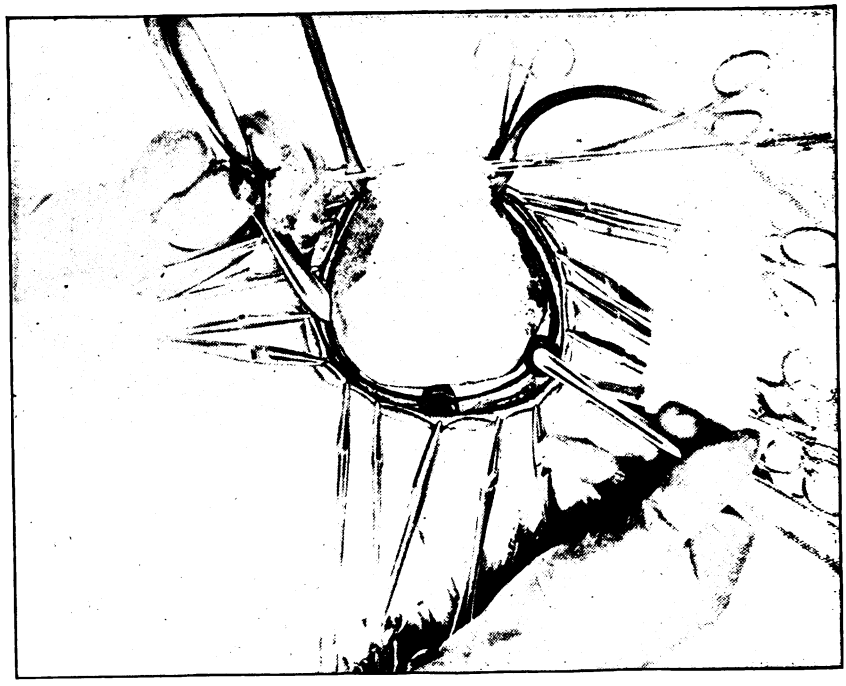

FIG. 111

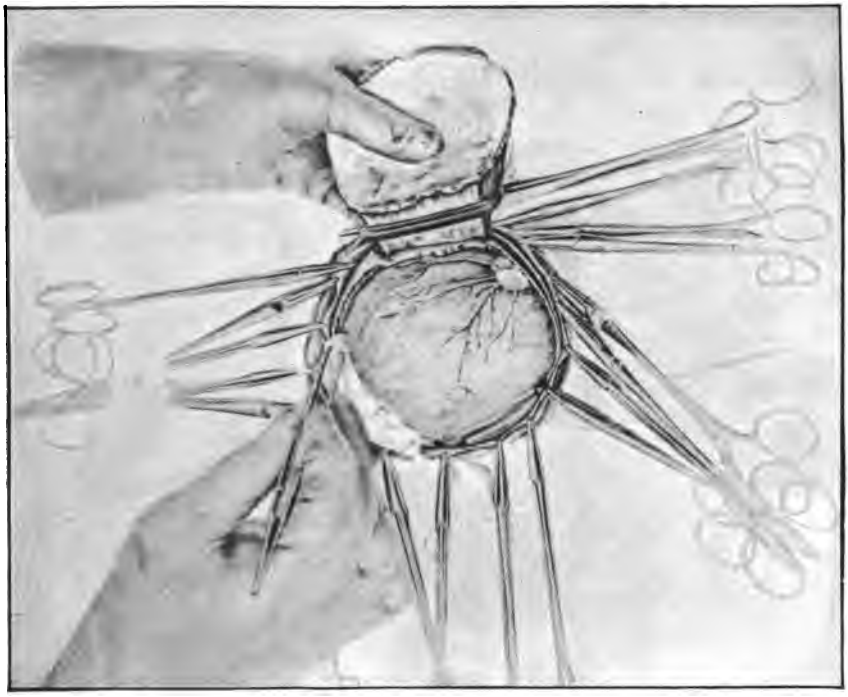

FIG. 112.

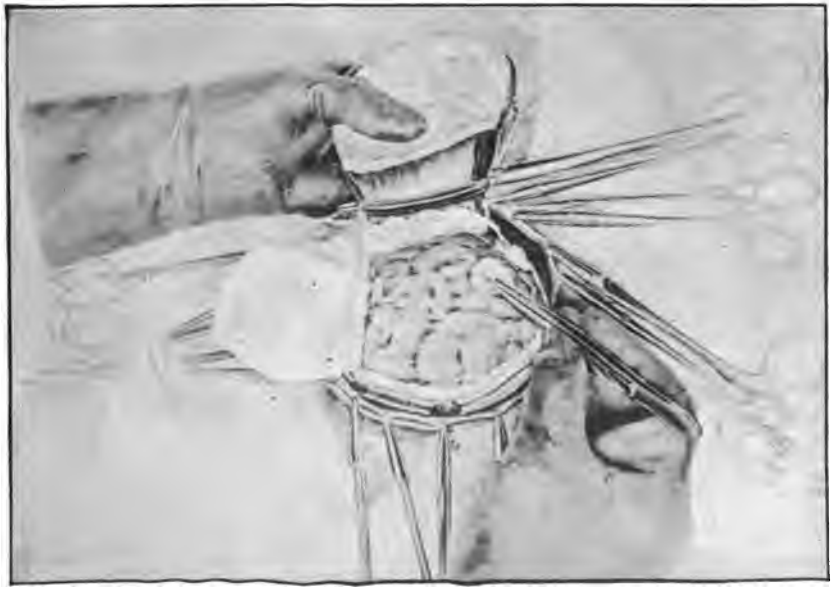

FIG 113

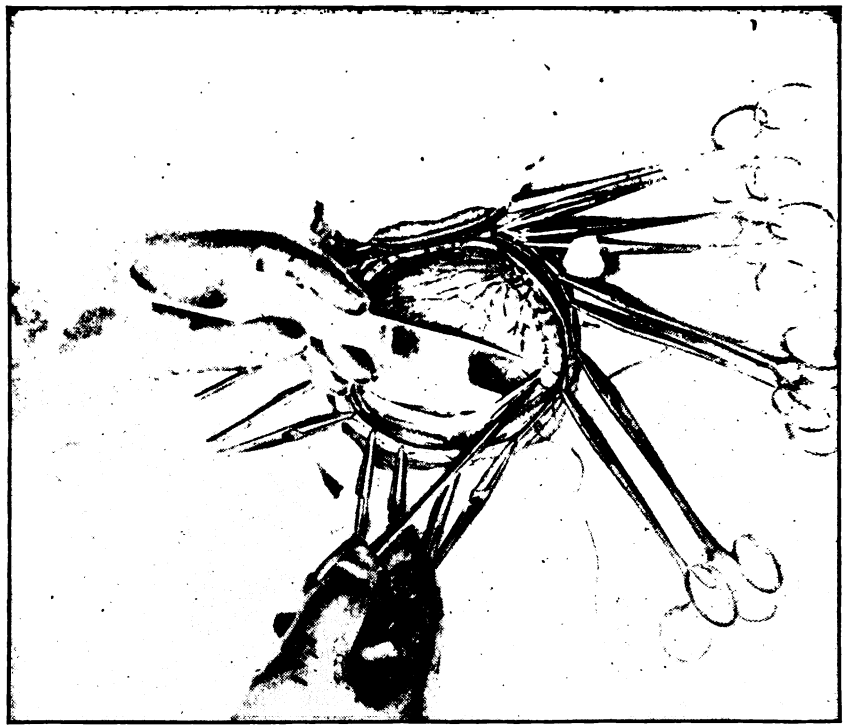

FIG. 114.

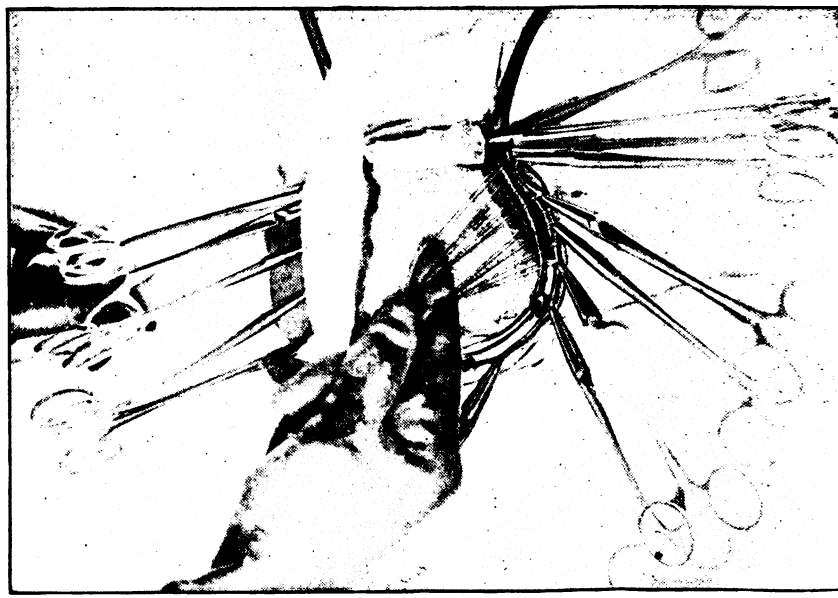

FIG. 115

as a wide exploration, a wide bone opening can be made by removing all the bone which is covered by temporal muscle.

The opening of the dura is made leaving a sufficient margin exposed for suturing. For decompressive purposes, the dura may be left open or excised over the temporal lobe.

After the operative procedure is completed, closure of the dural flap should be painstaking and accurate. Fine, interrupted sutures of silk are used.

The bone flap is then replaced and sutures of the same kind are used in closing the temporal muscle, fascia, and galea.

FIG. 116.

The skin closure is completed in the same way as described in the sub-temporal operation.

Steps of the suboccipital operation (From Tumors of the Nervus Acusticus: Cushing)—I. *Preparation of the patient.*—This consists in the mere omission of breakfast, the giving of an enema if necessary, and the shaving of the back of the patient's head the morning of the operation, over an area shown in the post-operative photographs accompanying the case reports. Proper shaving of the scalp is an art. In the case of women the hair should be parted in a curved line running from pinna to pinna and about 4 or 5 cm. above the line of the proposed incision. The hair above the line should be drawn up and gathered in a tight

braid which centers at the vertex, and the shaving should run downward from the exact line of the part.

Elaborate preparations for operation often defeat their own aims, in giving patients an uncomfortable night, in sending them to the operating room with their courage at a low ebb from intestinal cramps due to the unnecessary administration of a laxative and with the scalp sore from soap poultices or antiseptic applications. Some neurological surgeons have even advised a preliminary skin treatment lasting over a period of two days.

There are certain risks in a small area of shaving, but these are overcome by pinning the sheets securely into the scalp so that they remain immobile even through a long preparation. A little skill in the arrangement of their hair makes women presentable even after the shaving of the suboccipital area, and the advantages of this are not to be lost sight of.

II. *Position on the table.*—The operation being a symmetrical one, a face-down position is essential, and to make this position tolerable and permit of unimpeded free thoracic respiration, shoulder supports are necessary and the patient's face must rest in an outrigger beyond the table in such a way as to be accessible to the anesthetist. Some useful modifications in the crutch for the head have been devised by Grey, but to all intents and purposes it is much the same in principle as was originally described. The face rests in a well-padded horseshoe-shaped support which can be tilted or raised or moved in or out as necessary (Fig. 117). It is essential that the patients be comfortable and in such a position as to breathe freely, and this is one reason why they should always be put upon the table in the desired position before the anesthetic is started, for they may have to remain unmoved for several hours. The patient, moreover, should lie upon a thick mattress and not on a hard surface, and from personal tests it can be certified that the position is comfortable enough to favor sleep.

III. *The anesthetic.*—The most important factor is the anesthetic. No patient should be given inhalation narcosis by unskilled hands, but in these operations in particular, carried out on patients who have some deglutitory difficulty and whose respiratory center may readily become embarrassed, something more than the customary familiarity with giving an anesthetic is necessary.

Though de Martel advocates local anesthesia for cerebellar operations and others have employed various anesthetics or combinations of inhalation drugs, ether remains unquestionably the safest, though it admittedly is the most difficult to administer and it possesses the disadvantage of increasing the secretion of the cerebrospinal fluid and thus accentuating tension.

With the face-down position some method of giving ether vapor is necessary and we have come to rely on the Connell apparatus (Anesthetometer), by which warmed ether vapor is administered and the tension of the vapor is accurately recorded. Boothby has found that the same tension of the vapor appears to be required for the anesthetization of all vertebrates, large or small, and when one has learned to use the Connell machine, complicated as it appears, the giving of ether is not only greatly simplified but made a much more exact and even procedure. The face-down position, it may be added, is ideal for the effectual drainage of saliva and mucus.

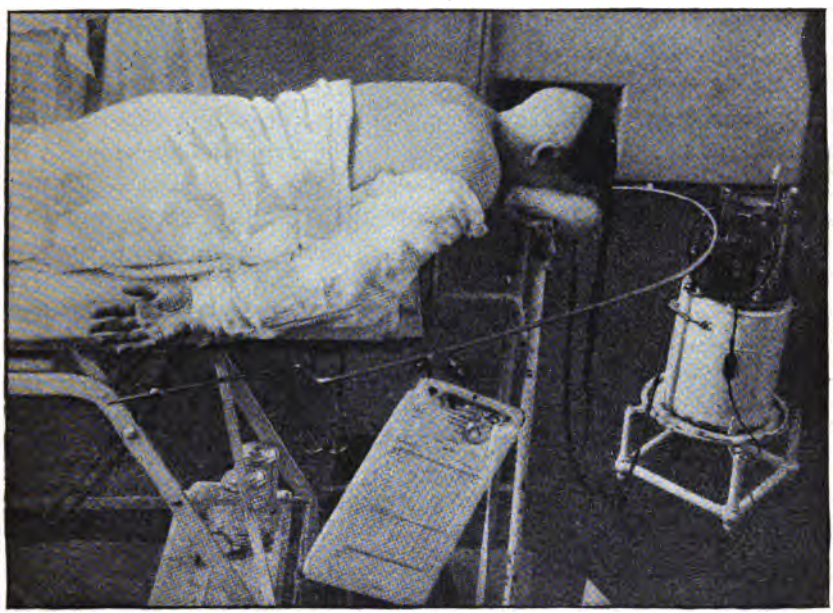

FIG. 117.—Patient in position before anesthetization

There are certain advantages in Meltzer's intratracheal anesthesia which are offset by the frequent difficulty of introducing the tube into the trachea and the fact that the complete relaxation of profound anesthesia is essential to the act. Were this introduction easier to accomplish and could the tube be accurately passed with the patient on his face, the method would be favored in all cerebellar operations in view of the possible need of artificial respiration. The face-down position, however, is the correct position for the employment of Schafer's method, and this has been relied upon in case of respiratory failure during the operation.

The anesthetist keeps an accurate ether chart, recording not only pulse rate. respiration, and ether tension but also by means of an

auscultatory method (Tycos) the systolic and diastolic pressures. Thus the patient's condition can be determined with almost the exactness of an animal whose pulse and respiration are recorded on a drum during a laboratory experiment. The anesthetized patient should be, and usually is, so quiet during these prolonged operations that the operator has no distractions whatsoever on that score, and though it would possibly appear that the keeping of such a record might put too heavy a burden upon the anesthetist, Keen has testified to the contrary in his "Ether Day Address."

IV. *Preparation of the field.*—The freshly shaved area of scalp (Fig. 117) over the occiput to the ears and back of the neck, is merely washed with green soap on a gauze sponge, followed by alcohol and bichlorid just as the patient is reaching the second stage of ether. Cushing deprecates the use of iodin, at present so popular for a skin preparation.

A cuticular scratch barely deep enough to draw blood is then made with the edge of the knife to outline the proposed incisions. The curvilinear line thus demarcated should run from mastoid to mastoid and arches possibly 4 cm. above the occipital protuberance. If one is reasonably certain as to the side of the lesion, as when an acoustic tumor is expected, the scratch on that side may be carried somewhat farther down on that side, so that the posterior margin of the mastoid is more accessible. The demarcation for the vertical incision should be made exactly in the midline, like the arrow in a drawn bow, and is carried downward 9 or 10 cm. possibly to the spine of the fifth cervical vertebra. Over the field a large layer of bichlorid gauze is then thrown.

A series of sheets are then applied. The first, a sheet with a semicircular notch with tapes at the angles, is draped over the wire hoop which serves to hold it away from the field and makes a tent for the anesthetist. The semicircle is pinned into the scalp just above the scratched line of incision and the tapes are tied by the anesthetist below the head crutch. Lateral towels are pinned to the side of the wire hoop, drawn taut, and, together with the semicircle of the first sheet, secured with a towel clip into the skin at the point of the mastoid, and where they cross are similarly secured to the skin in the midline of the lower neck. The instrument stand is moved up, and over all is thrown a gray sheet, which also has a semicircle cut in its margin and which encircles the field.

Every surgeon has his preference for the arrangement of his field of operation, and it may seem superfluous to go into this stage of an operation in much detail, but there must be no slipping nor changing of towels during such an operation in this circumscribed field, and the incision, to be exactly in the position desired, must be marked out before the cloths are placed, else it would be necessary

to leave uncovered anatomical landmarks which can not be cleaned beyond suspicion. It is a principle, I think, which could well be introduced into surgical operations elsewhere in the body.

V. *The making of the flaps.*—Every possible precaution should be taken to avoid loss of blood, and much blood can be lost from 10 or

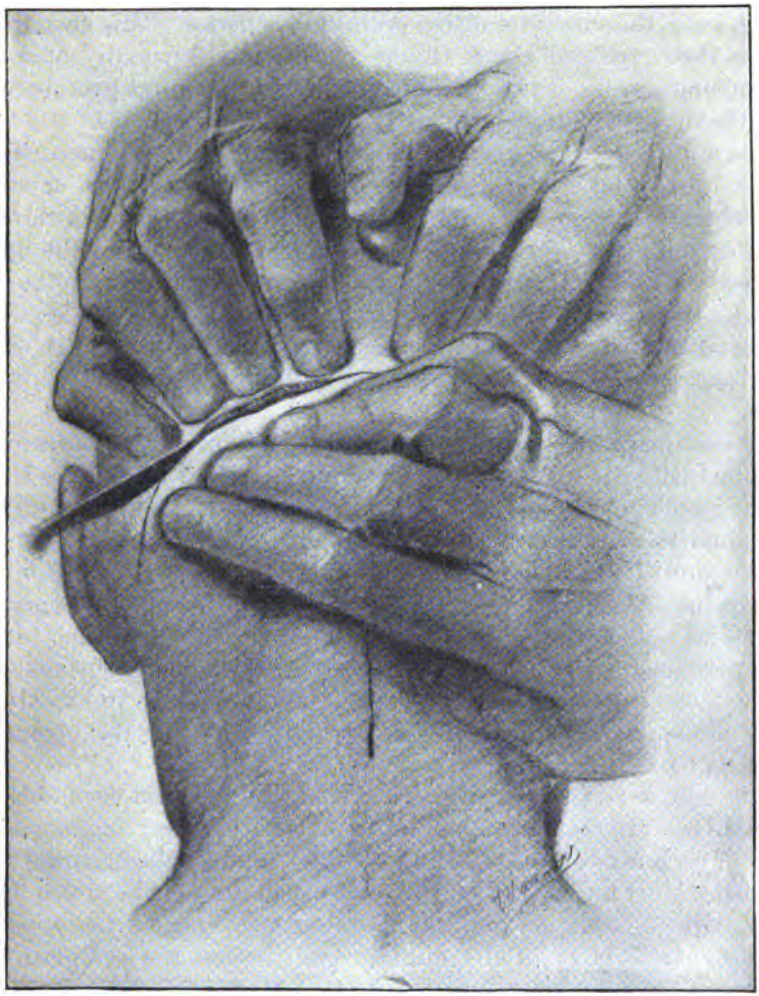

FIG. 118.—Showing method of controlling hemorrhage. Incision through galea on right half of wound.

12 inches of scalp incision, particularly when the extracranial vessels are distended, as they usually are in the presence of a brain tumor.

As described in a recent article upon the technic of a subtemporal decompression, we have come of late years to depend, except when a tourniquet can be used, solely upon the pressure of the fingers (Fig. 118) to control hemorrhage from scalp incisions, and with practised

assistants it is possible to make these incisions down through the galea and to separate the edges in the subaponeurotic layer with practically no bleeding whatsoever. Before the pressure is released a series of pointed clamps are attached to the galea, which is folded over the incision and effectually prevents subsequent bleeding. Particular care must be taken to catch the major suboccipital arteries which lie in the substance of the occipitalis muscles. This curvilinear flap is then "scalped" back (blunt dissection will usually suffice) in the subaponeurotic layer to slightly below the occipital protuberance and the muscular attachment of the superior curved line.

The midline incision (Fig. 119) is then made with like precautions, and it is essential that it be exactly in the midcervical line, or otherwise there will be unnecessary bleeding. It is carried down through the intermuscular spaces to the skull and to the spines of the upper cervical vertebræ, clamps being applied symmetrically on the two sides as the wound is deepened. This midline incision, made at this time and carried to the skull, serves to show the exact level at which the transverse cut should be made along the line of muscular attachment.

At this juncture a clamp of different pattern, so that later on it may be easily distinguished from the others, is attached on each side to the two upper edges of the vertical incision in the fascia (Fig. 119) and the fascia is divided along the superior curved line and drawn down. The muscle in turn is similarly treated after two clamps have been put at the angles, leaving just enough of a margin of fascial and muscular attachment to serve for subsequent suture. The placement of these clamps at the corners is an important step, for without these landmarks it will be found difficult to exactly reapproximate and resuture the layers at the close of the operation. (Fig. 123.)

The muscle is then scraped back from the bone on each side far enough to fully expose the margin of the foramen magnum, the edge of each mastoid process and well down under the occipital bone on either side, much farther than can be shown in the sketch (Fig. 120). During this act profuse bleeding may occur from emissary vessels, particularly in the midline and around the occipital protuberance, unless proper use is made of Horsley's bone wax, large quantities of which may sometimes be needed. Another place where embarrassing bleeding may occur is from the mastoid emissary, and here again wax is necessary or in difficult situations the placement of a fragment of muscle may be required.

VI. *The craniectomy.*—With a motor-driven burr one or more primary perforations are made over each hemisphere, and with rongeurs the openings are subsequently enlarged (Fig. 120) to the full extent

of the denuded area of bone. The lower margin of the lateral sinus is usually exposed on each side.

Difficulty may be experienced in crossing the midline, and in the removal of this thicker area of bone the placement of wax may be necessitated after each bite of the forceps. Needless to say, care

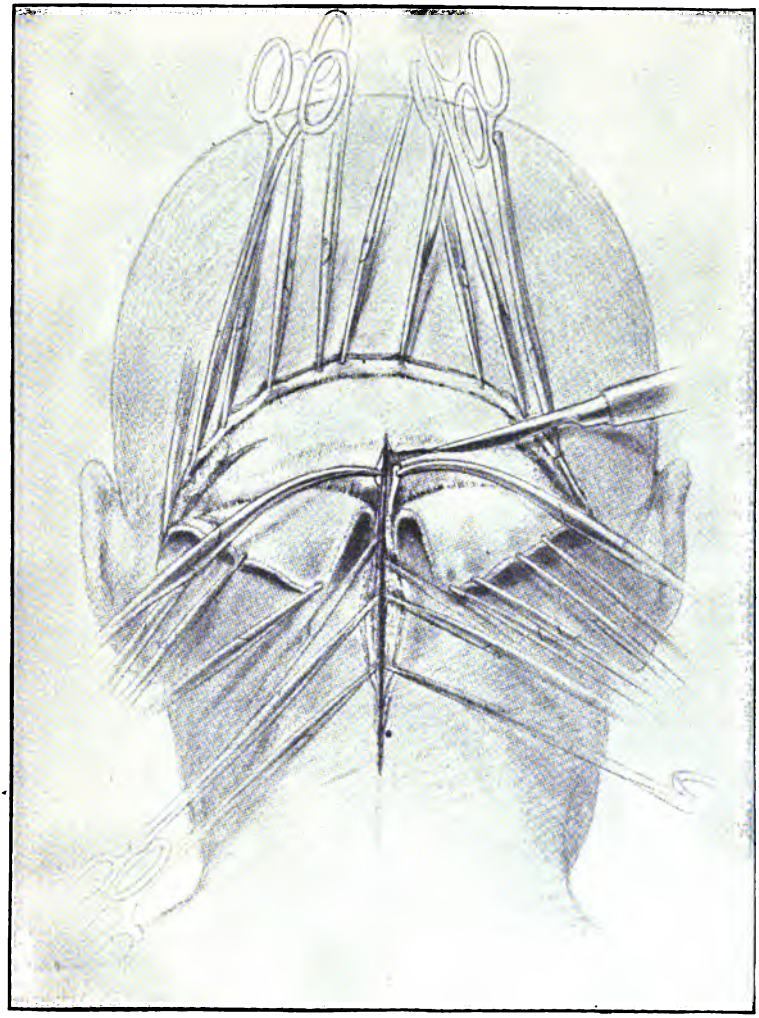

FIG. 119.—Mid-cervical incision carried to spine. Placement of identifying clamps on corners before lateral incisions are made through.

should be taken that the bone should be bitten outward, so as to avoid any jar of the forceps against the dura and brain. In the process of removing the posterior rim of the foramen and the bone to the side of it, emissary vessels from the sinus encircling the foramen may be encountered on each side, but they may be easily controlled.

In many cerebellar cases the bone will be found greatly thinned and is very easy to remove, and though this may be true also of recess tumors, it is perhaps less common than in the case of intracerebellar growths.

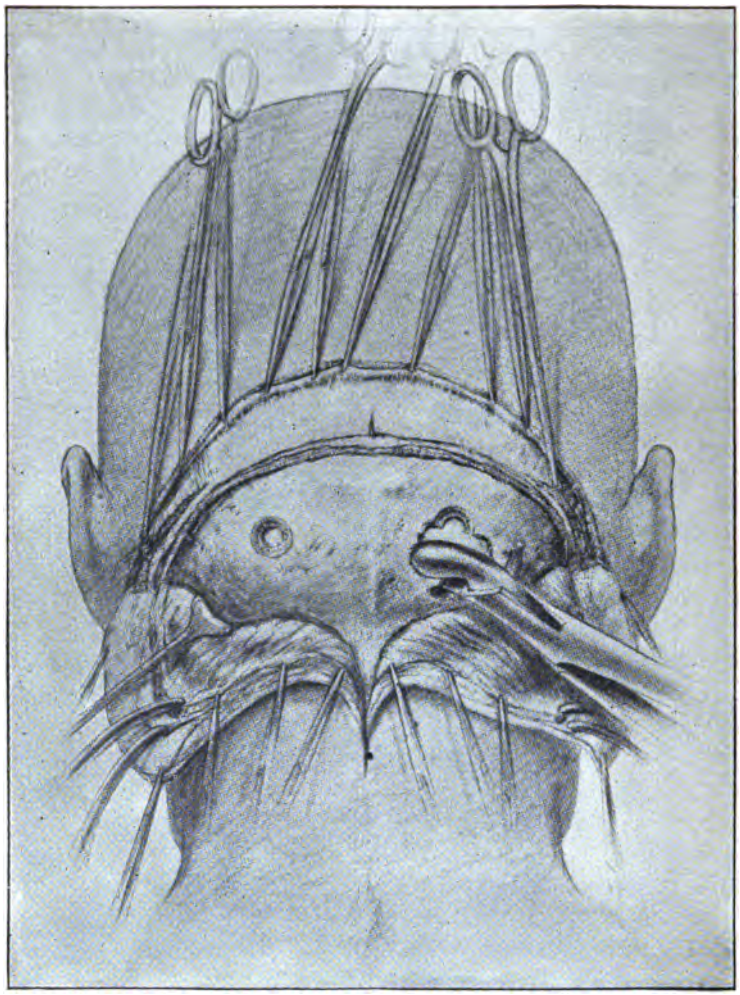

FIG. 120.—Showing denudation of suboccipital region; exposure of foraminal field by reflection of flaps; primary bone openings.

VII. *The dural opening and release of tension.*—The membrane will be found tense, and should an opening be made over one of the hemispheres before the tension has been lowered the cortex will protrude and is likely to become contused before the exposure can be completed. Consequently it is desirable to secure fluid at the outset. Hence a

minute primary dural opening (Fig. 121) is made low down near the foramen in order, if possible, to secure fluid from the posterior cistern. If there is a marked pressure cone and the cistern empty, cortex will immediately occlude the small opening, and it is far safer to immediately tap the lateral ventricle than to venture upon a further enlargement of the opening.

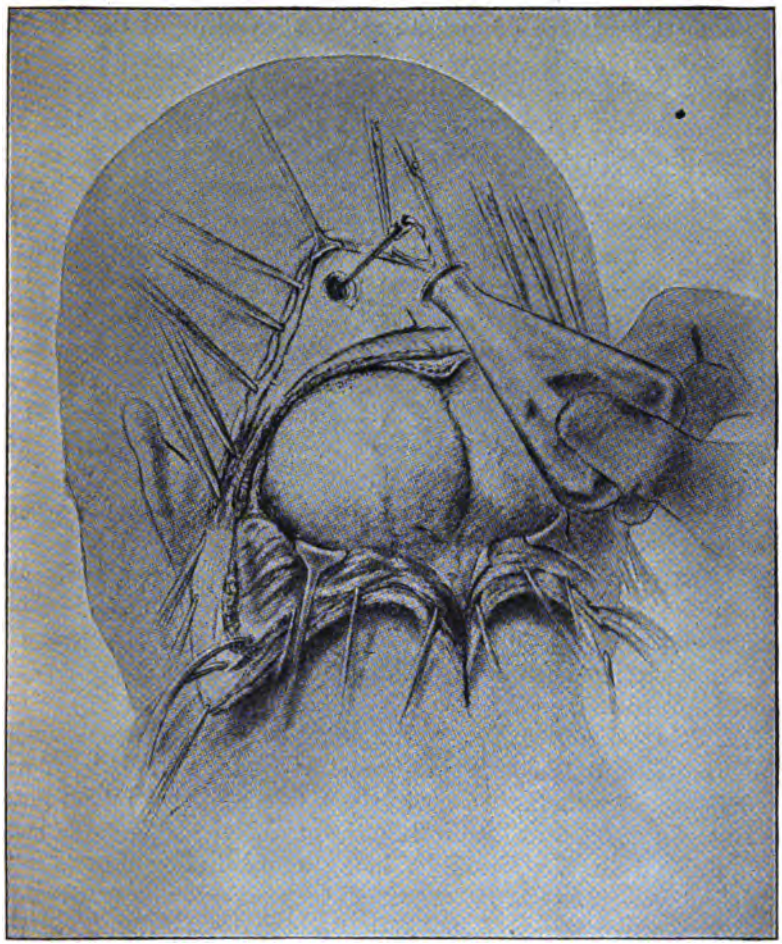

FIG. 121.—Showing, near the foraminal region, the primary small opening in the dura and subsequent puncture of the lateral ventricle if failure to secure fluid and extreme tension demand.

To tap the ventricle the scalp is drawn back in the subaponeurotic layer as shown in figure 121, and with a burr a perforation is made about 3 or 4 cm. above the superior curved line and 2 cm. from the midline. Through this opening the blunt cerebral aspirating needle is introduced slightly upward and outward, and with a good visualization of the ventricle the first tap rarely fails at a depth of 4 or 5

cm. to reach fluid. The needle is left in place during the remainder of the exploration and serves as a great safeguard against medullary pressure. Cushing has never seen any accident whatever from this procedure, which must have been carried out in over a hundred cases of cerebellar tumor. Indeed, on many occasions when hemorrhage from the extracranial tissue and the bone is particularly troublesome the intracranial tension has been thus lowered before the completion of the bone defect with immediate marked lessening of the tendency of the tissues to bleed.

After the ventricular tapping the dura will lose its tension and can be safely opened. Care must be taken in crossing the median line, for the sagittal cerebellar sinus may be of considerable size and the cerebellar falx may penetrate in some cases for a considerable distance. As the midline is crossed the cerebellum should be held away with a spoon spatula and the sinus may be caught, before division, with silver clips. Stellate incisions of the membrane to the margins of the bone defect are then made and the hemispheres fully exposed.

At this stage of the operation if there is any doubt of the diagnosis the two hemispheres should be compared for differences of tension, of form, or of vascularity. An intracerebellar cyst can usually be detected by palpation; an involvement of one hemisphere rather than the other by the displacement of the midline structures. Particular attention should be given to the conditions at the foramen, where a marked pressure cone may forebode ill.

An extensive foraminal herniation of the cerebellar tonsils (Fig. 122), however, is not so apt to accompany cerebellopontile lesions as intracerebellar tumors; indeed with some of the acoustic tumors in the series there has been but little displacement and a large posterior cistern is present.

VIII. *The wound closure.*—This stage of the operation, usually left to an assistant, is no less important and no less difficult, if properly done, than is the approach to the lesion. Indeed if the patient is to be given a sound neck and the post-operative leakage of cerebrospinal fluid from the wound is to be avoided, the most accurate closure in layers is necessary. In the average case it takes fully forty-five minutes to an hour to get in and as long to close, so that, with the allotment of time for the treatment of the lesion, many of these operations from the beginning of the anesthetization to the final closure approach or even exceed a three-hour period.

To take the strain off the flaps, the mass of clamps on each side is supported by the assistant who stands at the top of the head, and the four clamps, placed as described, on the corners of muscle and fascia are drawn up as shown in figure 123. The vertical incision is then closed accurately in several layers, three of which are indicated

in the sketch. Fine, waxed, black silk sutures passed on curved French needles are used throughout and tied three times so that they may be safely cut almost at the knot, thus avoiding any but the smallest amount of buried material.

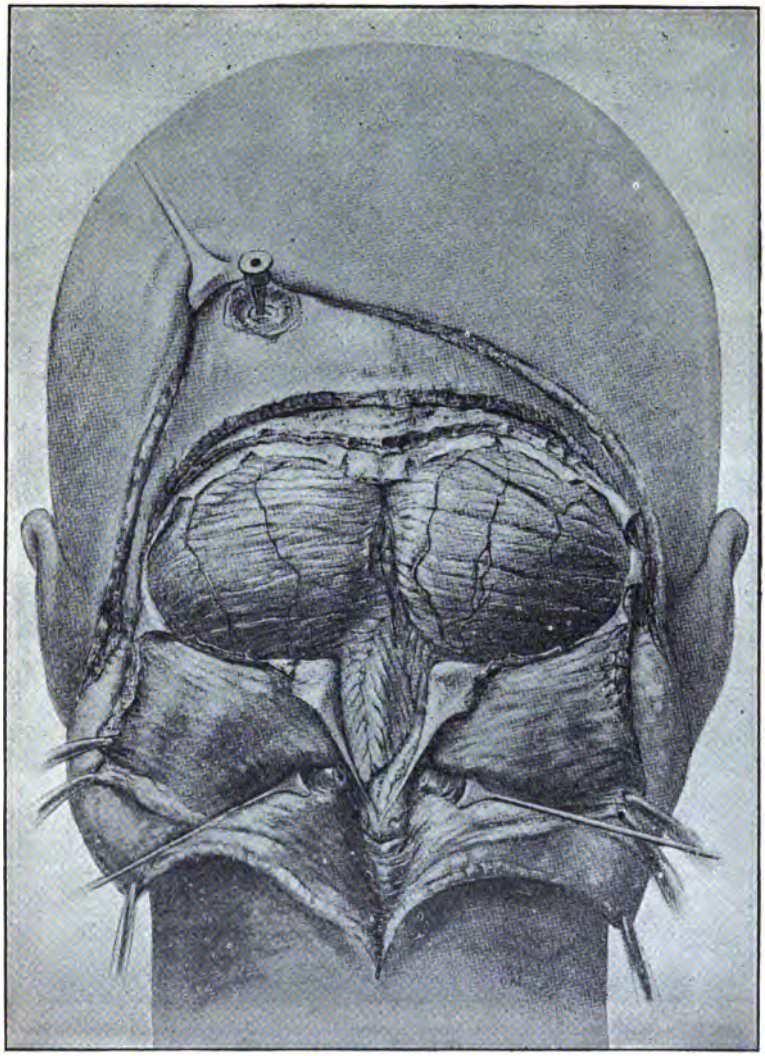

FIG. 122.—Showing extreme foraminal herniation (pressure cone) in a case in which tension was so great as to necessitate removal of the laminæ of the atlas and carrying the dural incision to the atlas before respiratory embarrassment was relieved.

The transverse incision is then sutured in turn, the crutch supporting the patient's head being raised and the neck extended slightly so as to facilitate bringing the edges together. A layer of interrupted sutures in the muscle is followed by a more secure second layer in the

fascia and a third layer to accurately approximate the galea. This layer not only catches all of the vessels of the scalp but holds the edges of the scalp so closely together that cutaneous sutures are only necessary in order to get an exact approximation of the skin edges. For this purpose fine round cambric needles are passed before tying,

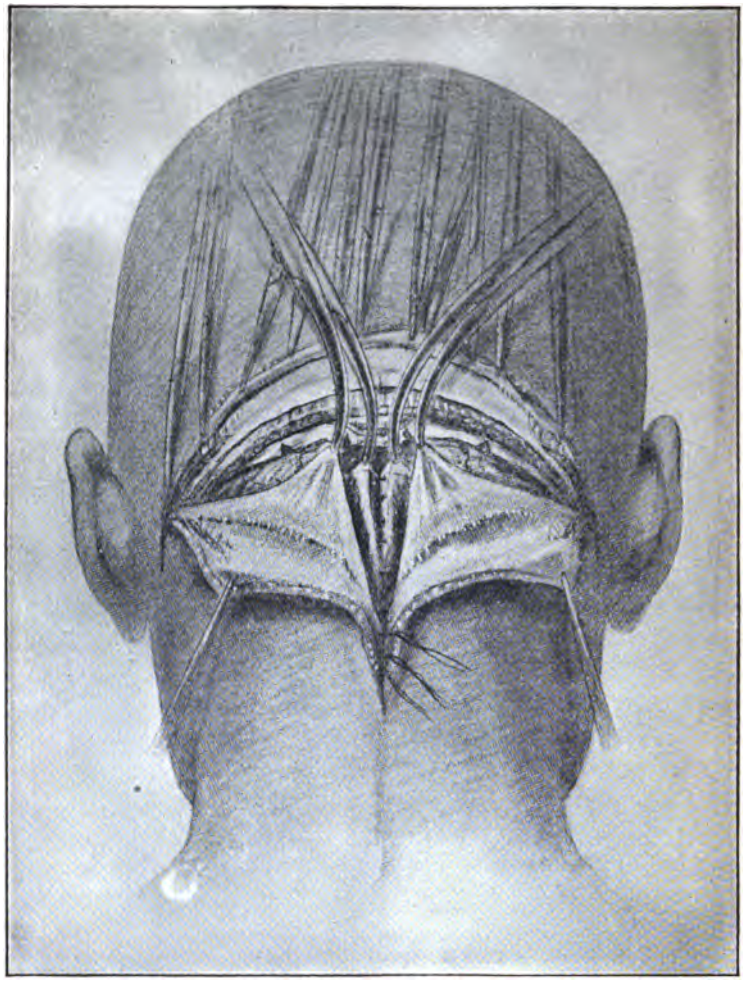

FIG. 123.—Showing early stage of closure of the median incision, the clamps identifying the corners of fascia and muscle being drawn up.

so that the wound edges may be "heaped up" and a broad approximation secured. Unquestionably a continuous suture is quicker, but it does not give equally exact union of the edges nor can it be left so long in the tissues, and the wound is of such magnitude and in such a position that it should be left until completely healed before the first dressing is made.

If precautions are not taken to have an absolutely secure closure, layer by layer, cerebrospinal fluid will find its way out, and though in one case in the series it got through the muscles under the scalp, in no instance has an actual leak occurred nor have I seen such an accident in any case of cerebellar operation for many years.

The different point of view of various clinics in regard to these wounds is best seen in the manner of closure and the importance placed upon it. Horsley, for example, always favored a loose closure of cranial wounds so that an escape of cerebrospinal fluid would occur.

Attention may be called to the fact that no vessels whatever are tied during the operation—in other words, no bits of tissue are strangulated by an encircling ligature. Such vessels as are divided in the extracranial tissues are occluded by the approximating sutures serially placed as the clamps are removed when the wound is closed. If the margin of the dura bleeds it is caught with silver clips, and if bleeding occurs from the nervous tissues themselves it is controlled by the placement of a fragment of raw muscle.

The minute black knots of the iron-dyed silk, it may be added, make very useful landmarks in case a subsequent exploration is necessary, for they remain practically unchanged for years and it is possible to retrace one's former steps by their aid on a second or a third session. These small knots of fine black silk are probably stronger for their size and are far better tolerated by the tissues than any other suture material, provided no tissue is strangulated in their placement.

Special emphasis should again be laid on the importance of a secure and separate closure of the galea by buried sutures in all cranial wounds which are to resist tension. Another purpose of this, in addition to security, is a cosmetic one. A scalp wound which is otherwise closed tends to part and to leave a broad ugly scar owing to destruction of the hair follicles.

CHAPTER VIII.

THE SPINAL CORD.

Anatomy.—The spinal cord is continuous above with the medulla oblongata, into which it passes without any sharp line of demarcation. Arbitrarily we take as the upper limit of the cord, the margin of the foramen magnum. From here the cord extends to the lower part of the first or the upper part of the second lumbar vertebra. In its lowermost portion it tapers off to form the conus medullaris, from the tip of which the filum terminale extends as a slender thread to the bottom of the vertebral canal. Below the end of the cord lies the cauda equina, consisting of a closely packed mass of nerve roots passing from their points of origin in the cord to their points of exit in the intervertebral foramina.

Throughout the thoracic region the spinal cord is more or less uniform in size. Above and below, in the cervical and lumbar region, it swells out to form the cervical and lumbar enlargements, from which come respectively the large nerve trunks for the upper and lower limbs. The cervical enlargement begins at the level of the third or fourth cervical vertebra, reaches its maximum opposite the fifth or sixth, and then gradually narrows until opposite the first or second dorsal spine it passes over into the typical appearance of the thoracic region.

The lumbar enlargement, which is smaller than the cervical, begins at the level of the tenth dorsal vertebra, reaches its maximum opposite the twelfth, and then tapers rapidly off to form the conus medullaris.

Leaving the cord in the antero-lateral region on either side, are the anterior root fibers. They pass laterally toward the dura, combining to form the anterior roots. Similarly, the posterior roots are formed by fibers arising from the cord at its postero-lateral aspect. The two roots pierce the dura separately, and join to form the spinal nerve. On the posterior root, just after it pierces the dura, lies the spinal ganglion, or posterior root ganglion.

From the entire length of the spinal cord there arises a continuous series of nerve roots, symmetrically arranged on the two sides, and combining to form a series of spinal nerves. Each of these nerves passes out through an intervertebral foramen, in a certain fixed relation to the vertebral column. In the dorsal, lumbar, and sacral region, each pair of nerves passes out below the correspondingly named vertebra, the first dorsal nerve below the first dorsal

vertebra, the first lumbar nerve below the first lumbar vertebra, etc. In the cervical region there are eight cervical nerves and only seven cervical vertebræ. The first pair leaves the spinal canal between the occipital bone and the atlas; and so the first seven cervical nerves pass out above the correspondingly named vertebræ. The eighth cervical emerges below the seventh cervical vertebra.

That portion of the spinal cord which gives rise to a pair of spinal nerves is called a segment of the cord, and takes its name from the pair of nerves to which it gives origin. There are eight cervical, twelve dorsal, five lumbar, and five sacral segments.

In early fetal life, the spinal cord extends to the lower end of the spinal canal. In a general way, each cord segment lies opposite a like-named vertebra, and the spinal nerves pass directly outward to their foramina of exit. In the course of development, the vertebral column grows out of all proportion to the growth of the cord, so that in adult life the lower end of the cord (the sacral segments) comes to lie opposite the lower body of the first lumbar vertebra. The change in relationship is relatively slight in the cervical region, but shows a progressive increase in dorsal, lumbar, and sacral regions. This results in a change in the intradural course of the spinal nerves, especially the lower ones. In the cervical region they continue to run almost horizontally outward to their foramina of exit. In the lower cervical and dorsal region the course becomes somewhat oblique. The obliquity increases progressively in the lower dorsal and lumbar region, and finally, in the lower lumbar and sacral region the roots run almost vertically downward to their foramina of exit. The roots from the lower lumbar and sacral regions, descending below the end of the cord, form the cauda equina. The relationship between cord segment, spinal roots, and vertebral body is shown in the diagram (Fig. 124). It will be noticed that in the sacral region the segments are narrow, and the roots come off in close proximity to each other.

Spinal membranes.—As in the case of the brain, the spinal cord has three coverings, the dura mater, the arachnoid, and the pia mater. The dura is a thick, tough membrane. It is perforated laterally for the passage of the anterior and posterior spinal roots, and sends out a lateral prolongation which envelopes each of the spinal roots as it emerges. The dura is not in immediate contact with the periosteum which lines the bony wall of the spinal canal, but is separated from it by a relatively thick layer of loose, fatty tissue, with a rich vascular supply and especially with extensive venous plexuses.

Closely applied to the inner surface of the dura is the delicate arachnoid membrane. It is connected with the pia by fine strands and bands of tissue, but is separated from it by the spinal fluid which fills the subarachnoid space. As in the case of the dura, the arach-

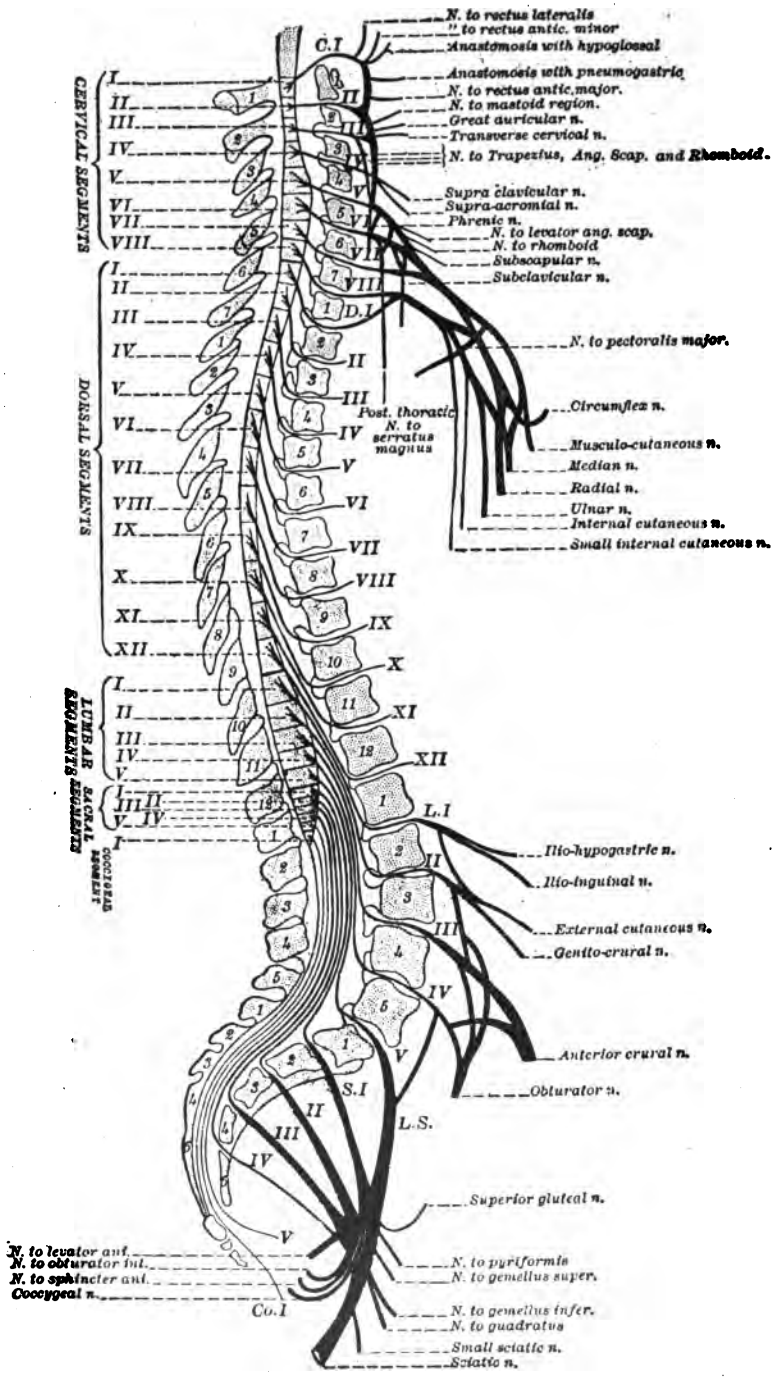

FIG. 124.—The relation of the segments of the spinal cord and their nerve roots to the bodies and spines of the vertebræ. (Dejerine et Thomas, Mal. d. l. Moelle Epiniere, Paris, 1902.)

noid sends out a prolongation which invests each of the emerging spinal nerves.

The pia is a delicate membrane which intimately invests the cord, forms septal prolongations into it, and dips into the anterior median fissure to which it carries the blood vessels.

Passing from the pia on the lateral aspect of the cord through its entire length is the fibrous band of the dentate ligament. It arises from the pia between the anterior and posterior roots and is inserted into the inner surface of the dura by numerous slips or dentations. It terminates below in a two-pronged fork, at the level of the first lumbar vertebra. According to Elsberg, the first lumbar root rests upon the inner prong of the fork, which thus becomes an important anatomical landmark for identifying that root in the course of surgical procedures.

Blood supply.—The vertebrals give off the anterior and posterior spinal arteries, which run down the anterior and posterior aspects of the cord. From the intercostals, lumbar and sacral arteries, small branches reach the cord by way of the spinal roots, and form a vascular ring about the cord. From the anterior spinal artery, which continues down in the anterior median fissure, branches pass off which supply especially the anterior horns. From the vascular ring surrounding the cord small vessels pass inward for the supply of the white matter and the posterior horns.

Histological structure.—In order to understand the symptomatology of disease and injury of the spinal cord, a brief description of its histological structure and relationships is necessary. While varying considerably at different levels, a general structural character is maintained throughout.

The cord is divided into two lateral halves separated ventrally by the anterior median fissure, a relatively broad, shallow groove, and dorsally by the posterior median septum—an inward prolongation of the pia. The two halves are connected by the gray commissure and the white commissure.

Each half of the cord contains a central core of gray matter. These are symmetrical on the two sides, and are connected by the gray commissure. Dorsally, a band of gray matter runs backward and outward to the surface of the cord. Taken as a whole, this mass of gray matter is usually described as being the shape of a butterfly. The anterior portions of gray matter are called the anterior horns; the posterior prolongations, the posterior horns. Within the gray commissure lies the small central canal.

Surrounding the core of gray matter everywhere, excepting at the tip of the posterior horns, is the white matter of the cord. It, too, is symmetrically distributed on the two sides, and connected in the mid-line, at the base of the anterior median fissure, by a narrow strand of fibers, the white commissure.

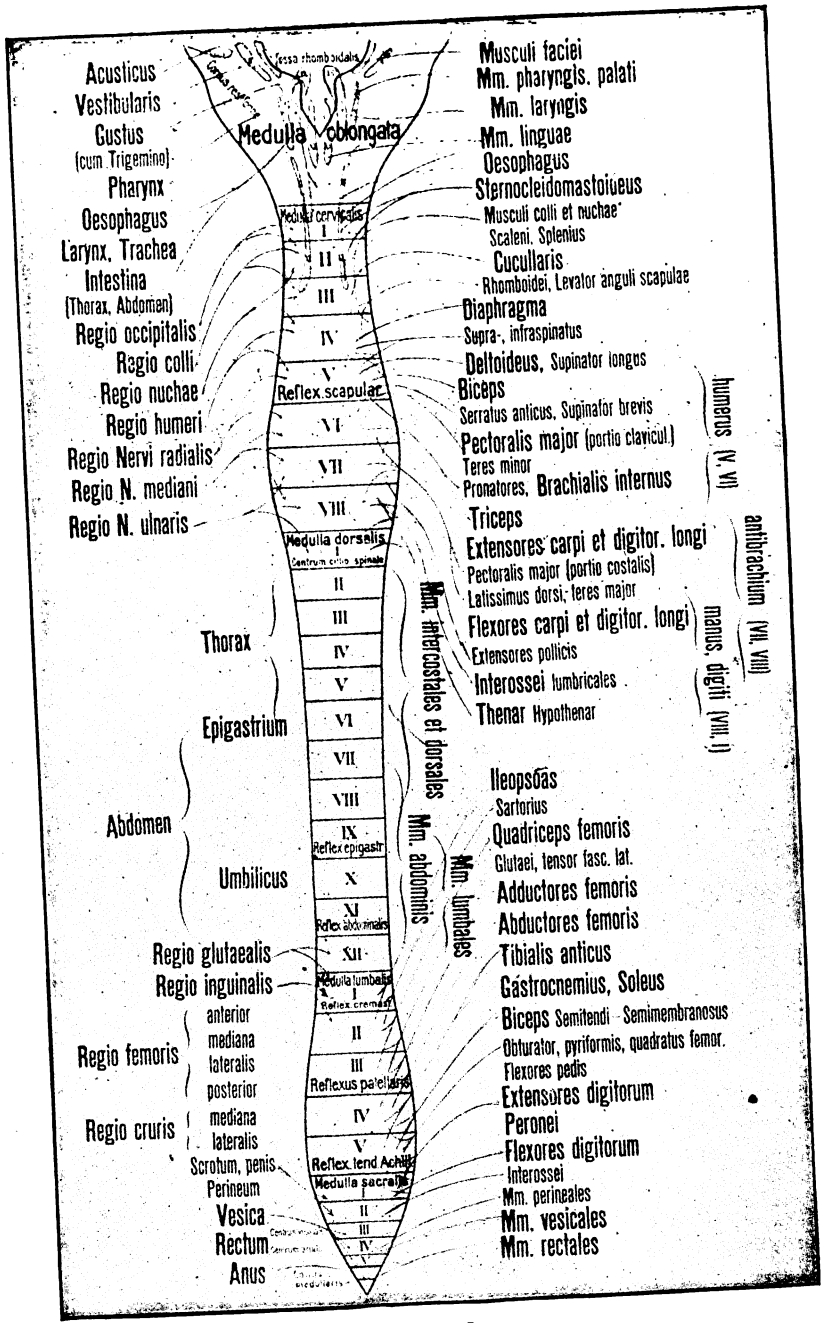
Acusticus
Vestibularis
Gustus
(cum Trigemino)
Pharynx
Oesophagus
Larynx, Trachea
Intestina
(Thorax, Abdomen)
Regio occipitalis
Regio colli
Regio nuchae
Regio humeri
Regio Nervi radialis
Regio N. mediani
Regio N. ulnaris
Thorax
Epigastrium
Abdomen
Umbilicus
Regio glutaealis
Regio inguinalis
Regio femoris
anterior
mediana
lateralis
posterior
Regio cruris
mediana
lateralis
Scrotum, penis
Perineum
Vesica
Rectum
Anus
Fossa rhomboidalis
Medulla oblongata
Medulla cervicalis
Reflex scapular
Medulla dorsalis
Reflex epigastr.
Reflex abdominalis
Medulla lumbalis
Reflex cremast.
Reflexus patellaris
Reflex tend. Achill.
Medulla sacralis
Musculi faciei
Mm. pharyngis. palati
Mm. laryngis
Mm. linguae
Oesophagus
Sternocleidomastoideus
Musculi colli et nuchae
Scaleni. Splenius
Cucullaris
Rhomboidei, Levator anguli scapulae
Diaphragma
Supra-, infraspinatus
Deltoideus, Supinator longus
Biceps
Serratus anticus. Supinator brevis
Pectoralis major (portio clavicul.)
Teres minor
Pronatores. Brachialis internus
Triceps
Extensores carpi et digitor. longi
Pectoralis major (portio costalis)
Latissimus dorsi, teres major
Flexores carpi et digitor. longi
Extensores pollicis
Interossei lumbricales
Thenar Hypothenar
humerus (V. VI)
antibrachium (VII. VIII)
manus, digiti (VIII. I)
Mm. intercostales et dorsales
Mm. abdominis
Mm. lumbales
Ileopsoas
Sartorius
Quadriceps femoris
Glutaei, tensor fasc. lat.
Adductores femoris
Abductores femoris
Tibialis anticus
Gastrocnemius, Soleus
Biceps Semitendi Semimembranosus
Obturator, pyriformis, quadratus femor.
Flexores pedis
Extensores digitorum
Peronei
Flexores digitorum
Interossei
Mm. perineales
Mm. vesicales
Mm. rectales

FIG. 125.

The difference in gross appearance of gray and white matter depends on differences of histological structure. The gray matter contains nerve cells, non-medullated nerve fibers, neuroglia, and a rich supply of capillaries. The white matter gains its characteristic appearance from the closely packed masses of medullated nerve fibers, with some neuroglial tissue and a much smaller capillary supply.

Functions.—The spinal cord has two chief functions: first, it is the reflex center for all activities of the trunk and limbs; second, it forms the conduction path between these lower centers and the brain.

The essential elements of the cord as a reflex center lie in the nerve cells of the gray matter. In the anterior horns are found groups of large, multipolar nerve cells—the motor cells of the cord. Each of these gives rise to an axon which leaves the cord by the anterior or motor root, and passes by way of a peripheral nerve to the muscle fiber which it innervates. A group of such nerve cells with their axons combine for the supply of a muscle. The supply for any given muscle is not confined to a single cross section of the cord, but extends in a longitudinal band, up and down the cord over a region of two, three or even more segments. Each muscle, then, is represented, in several segments of the cord; and these segments are always the same (although with some variation) for a given muscle. Fig. 125 shows a diagram of the cord, with the segmental representation of each muscle. The following table gives a list of the more important muscles of the body, with the segments of the cord in which they are represented:

TABLE V.—*List of the more important muscles of the body, with the segments of the cord in which they are represented.*

	Segment.	Nerve.	Action.
MUSCLES OF BACK.			
Trapezius	C2–4	C2–4 and spinal accessory.	Rotation of scapula, raises shoulder, moves head to side.
Latissmus dorsi	C6–8	Long subscapular	Adduction, inward rotation and extension of arm.
Rhomboids	C5	Dorsalis scapulæ	Rotates and moves scapula backward.
MUSCLES OF NECK, CHEST, AND ABDOMEN.			
Sternomastoid	C2–3	C2–3 and spinal accessory.	Rotates and draws head to shoulder.
Pectoralis major:			
Clavicular portion	C5–6	Ext. and int. ant. thoracic.	Adduction of arm; also downward and forward movement of arm.
Costal portion	C7–8		
Serratus anticus	C5–7	Long thoracic	Fixes scapula; rotates scapula forward.
Diaphragm	C3–5	Phrenic	Respiration.
Rectus abdominis	D5–12	Intercostals 5–12	Abdominal muscles.
Obliquus externus	D5–12	Intercostals 5–12	
Obliquus internus	D8–L1	Intercostals 8–12 and L1.	
Transversalis	D7–L1	Intercostals 7–12 and L1.	
MUSCLES OF EXTREMITIES.			
Upper extremities:			
Shoulder—			
Deltoid	C5–6	Circumflex	Abduct arm.
Spinatus	C5–6	Suprascapular	Outward rotation and abduction of arm.
Teres major	C(5)–6–(7)	Long subscapular	Inward rotation of arm.

TABLE V.—*List of the more important muscles of the body, etc.*—Continued.

	Segment.	Nerve.	Action.
MUSCLES OF EXTREMITIES—con.			
Upper extremities—Continued.			
Arm—			
Biceps	C5-6	Musculocutaneous	Flexes forearm and supinates.
Brachialis anticus	C5-6	Musculocutaneous	Flexes forearm.
Triceps	C6-7-(8)	Musculospiral	Extends forearm.
Forearm—			
Supinator longus	C5-6	Musculospiral	Flexes forearm.
Pronator radii teres	C6-7	Median	Pronates forearm.
Flexor carpi radialis	C6-7	Median	Flex wrist.
Palmaris longus	C(7)-8-(D1)	Median	
Flexor carpi ulnaris	C(7)-8-(D1)	Ulnar	
Flexor sublimis digitorum.	C7-8, D1	Median	Flexes middle phalanges 2-5 fingers.
Flexor profundus digitorum.	C7-8, D1	Median and ulnar	Flexes terminal phalanges 2-5 fingers.
Flexor longus pollicis	C6-7	Median	Flexes last phalanx of thumb.
Pronator quadratus	C6-8, D1	Median	Pronates forearm.
Extensor carpi radialis	C(5)-6-7	Musculospiral	Extend wrist.
Extensor carpi ulnaris	C(6)-7-8	Musculospiral	
Extensor communis dig.	C6-8	Musculospiral	Extend proximal phalanges.
Extensor indicis	C6-8	Musculospiral	
Extensor minimi dig.	C6-8	Musculospiral	
Abductor longus pollicis	C6-7	Musculospiral	Abducts thumb.
Extensor brevis pollicis	C6-7	Musculospiral	Extends thumb at proximal phalanx.
Extensor longus pollicis	C6-7-(8)	Musculospiral	Extends thumb at terminal phalanx and abducts thumb.
Hand—			
Adductor pollicis brevis	C6-7	Median	Abducts thumb.
Flexor brevis pollicis	C6-7	Median and ulnar	Flexes thumb.
Opponens pollicis	C6-7	Median	Apposition of thumb.
Abductor pollicis	C6-7	Ulnar	Adducts thumb.
Abductor minimi dig.	C8-D1	Ulnar	Abducts little finger.
Flexor brevis minimi dig.	C(7)-8-(D1)	Ulnar	Abducts and flexes little finger.
Opponens minimi dig.	C(7)-8-(D1)	Ulnar	Draws forward little finger.
Lumbricals	C7-8-(D1)	Median and ulnar	Abduct and adduct fingers.
Interossei	C7-8-(D1)	Ulnar	Flexion of proximal phalanges, extension of distalphalanges. Abduct and adduct phalanges.
LOWER EXTREMITIES.			
Hip			
Iliacus	L2-4	Lumbar plexus and ant. crural.	Flexes thigh.
Psoas major and minor	L1-3-(4)		
Gluteus maximus	L(4)-5-S1-(2)	Inferior gluteal	Extends thigh.
Tensor fasciæ latæ	L4-5	Superior gluteal	Flexes thigh.
Gluteus medius and minimus	L4-5.S1	Superior gluteal	Abducts thigh and rotates thigh in.
Quadratus femoris	L4-5. S1	Lumbar plexus	Rotates thigh out.
Thigh:			
Sartorius	L2-3	Anterior crural	Rotates leg in; flexes thigh and leg.
Quadriceps femoris	L2-4	Anterior crural	Extends leg.
Gracilis	L2-4	Obturator	Adducts thigh.
Adductors—longus, brevis, magnus.	L2-4	Obturator (additional fibers from ant. crural and sciatic.	Adduct thigh.
Biceps	L(4)5. S1-2	Sciatic	Flex leg.
Semimembranosus	L4-5. S1		
Semitendinosus	L4-5. S1		
Leg:			
Tibialis anticus	L4-(5)	Anterior tibial	Dorsal flexion and supination of foot.
Extensor longus digitorum	L4-5S1	Anterior tibial	Dorsal flexion of toes and foot.
Extensor longus pollicis	L4-5-(S1)	Anterior tibial	Dorsal flexion great toe.
Peronei—longus, brevis, tertius.	L5. S1	Musculocutaneous	Pronation of foot.
Gastrocnemius	L(4). 5. S1-2	Internal popliteal	Flexion of foot.
Soleus	L(4). 5. S1-(2)		
Tibialis posticus	L5. S1-(2)	Posterior tibial	Adducts foot.
Flexor longus digitorum	L5-S1-2	Posterior tibial	Flexes toes.
Flexor longus pollicis	L5-S1-2	Posterior tibial	Flexes great toe.
Foot:			
Extensor brevis pollicis	L4-5(S1)	Anterior tibial	Extends great toe.
Extensor brevis digitorum	L4-5-S1	Anterior tibial	Extends toes.
All other small muscles of foot (muscles of sole, interossei, etc.)	L5-S1-2	Plantar	Movement of toes.

In the posterior horns lie the small cells about which terminate many of the afferent fibers which enter the cord by way of the posterior roots. These roots have their origin in the spinal ganglia, lying just outside the dura. The ganglia consist of masses of nerve cells, each giving origin to a single axon. The axon divides in T-shape, one branch running to the periphery and joining with similar fibers to form the peripheral sensory nerve, and the other entering the cord as part of the posterior spinal root.

For each segment of the cord there is a pair of posterior roots, one on each side. Each of these roots assembles the nerve fibers representing a given skin area or segment of the body. Figure 126 shows the particular skin area presided over by each spinal root and the corresponding segment of the cord.

It is thus seen that each muscle and each skin area of the body has its representation in some special region of the spinal cord, and by referring a motor or sensory disturbance to the segment of the cord which controls it, we are able to localize spinal cord lesions.

As was stated, the essential apparatus for the reflex activities of the spinal cord is contained in the gray matter, more particularly in the cells of the posterior horn and the anterior horn. Every reflex act consists of an afferent (sensory) impulse and an efferent (motor) impulse. A sensory impulse is conveyed from the periphery by way of peripheral sensory nerve, posterior root ganglion and posterior root, to the cells of the posterior horn. From here it is transferred to the cells of the anterior horn. Here it becomes a motor impulse, and passes out of the cord by way of the anterior root and peripheral motor nerve, to the appropriate muscle.

Muscle reflexes.—It can be readily seen that the simplest reflex arc consists in the response which is obtained from irritation of a single muscle and that the tendon reflexes represent the responses of a number of muscles. Tapping the body of the rectus femoris produces a contraction of that muscle alone, whereas tapping of the patellar tendon produces a reponse of a great number of muscles.

These muscle reflexes are just as important as the tendon reflexes. They are obtained by tapping the body of the muscle itself. For example, in the chest and abdomen, tapping any part of the muscle will produce a response which can be readily appreciated in a thin individual. It is more difficult to obtain in fat persons. In such diseases as poliomyelitis or in destructive lesions of the cells of the anterior horn, the muscle reflexes are absent. Consequently, the absence of a muscle reflex indicates interference with its particular arc and is an accurate indication of the level of the lesion.

Normal muscle tone depends on the integrity of this spinal reflex arc; any break in the arc, whether of the afferent limb or of

the efferent limb, results in loss of the tone. The same is true of the tendon reflexes; these are likewise dependant on the integrity of the spinal reflex arc, and any interference with the arc causes reduction or loss of the reflex. Each tendon jerk is represented in a particular region of the cord, depending on the area from which the afferent impulse comes, and the particular muscles engaged in the motor response. The following table gives the segmental representation of the more important of the reflexes:

TABLE VI.—*The centers for the skin and tendon reflexes in the spinal cord (Bing).*

Tendon reflex.	Skin reflex.	Elicited by—	Result.	Localization
	Scapular reflex....	Irritation of skin over scapula.	Contraction of muscles of shoulder blade.	C5-D1.
Biceps reflex.....	.....	Blow on tendon of biceps.	Flexion of forearm.....	C5-6.
Triceps reflex.....	.....	Blow on tendon of triceps.	Extension of forearm.....	C6-7.
Scapulo-humeral reflex.	.....	Blow on lower inner angle of scapula.	Adduction of arm.....	C6-7.
Radius reflex.....	.....	Blow on styloid process of radius.	Supination of forearm....	C7-8.
	Palmar reflex.....	Irritation of palm of hand.	Flexion of fingers.....	C8-D1
	Epigastric reflex...	Irritation of lower part of chest.	Contraction of epigastric region.	D7-9.
	Upper abdominal reflex.	Irritation of skin over upper abdomen.	Contraction of abdominal muscles.	D8-9.
	Lower abdominal reflex.	Irritation of skin of lower abdomen.	.....do.....	D10-12.
	Cremasteric reflex.	Irritation of region over adductor muscles.	Elevation of testis.....	L1-2.
Patellar reflex.....	.....	Blow on tendon of quadriceps.	Extension of leg.....	L2-4.
	Gluteal reflex.....	Irritation of skin of gluteal region.	Contraction of gluteal muscles.	L4-5.
Achilles reflex.....	.....	Blow on Achilles tendon.	Extension of foot.....	S1-2.
	Plantar reflex.....	Irritation of skin of sole..	Flexion of toes.....	S1-2.
	Anal reflex.....	Pricking of perineum.....	Contraction of sphincter ani.	S5.

The second function of the cord, that of connecting the lower (reflex) centers with the higher (cerebral) centers, is subserved chiefly by the white matter. Its compact fiber bundles contain the conducting pathways leading up or down. There are several of these pathways or tracts, each with a more or less distinct function. Normally there is no line of demarcation to separate them from each other; but, by means of Wallerian or secondary degeneration—that degeneration which occurs in a nerve fiber after it has been separated from its cell of origin—it has been possible to localize and delimit with more or less accuracy a number of the important paths. We will mention only the most important.

A. DESCENDING (MOTOR) TRACTS.

I. *Lateral pyramidal tract (crossed pyramidal tract.)*—This is a part of the great cortico-spinal projection system. The cells of origin lie in the precentral convolution. The fibers descend through the internal capsule, decussate at the upper limit of the cord, and de-

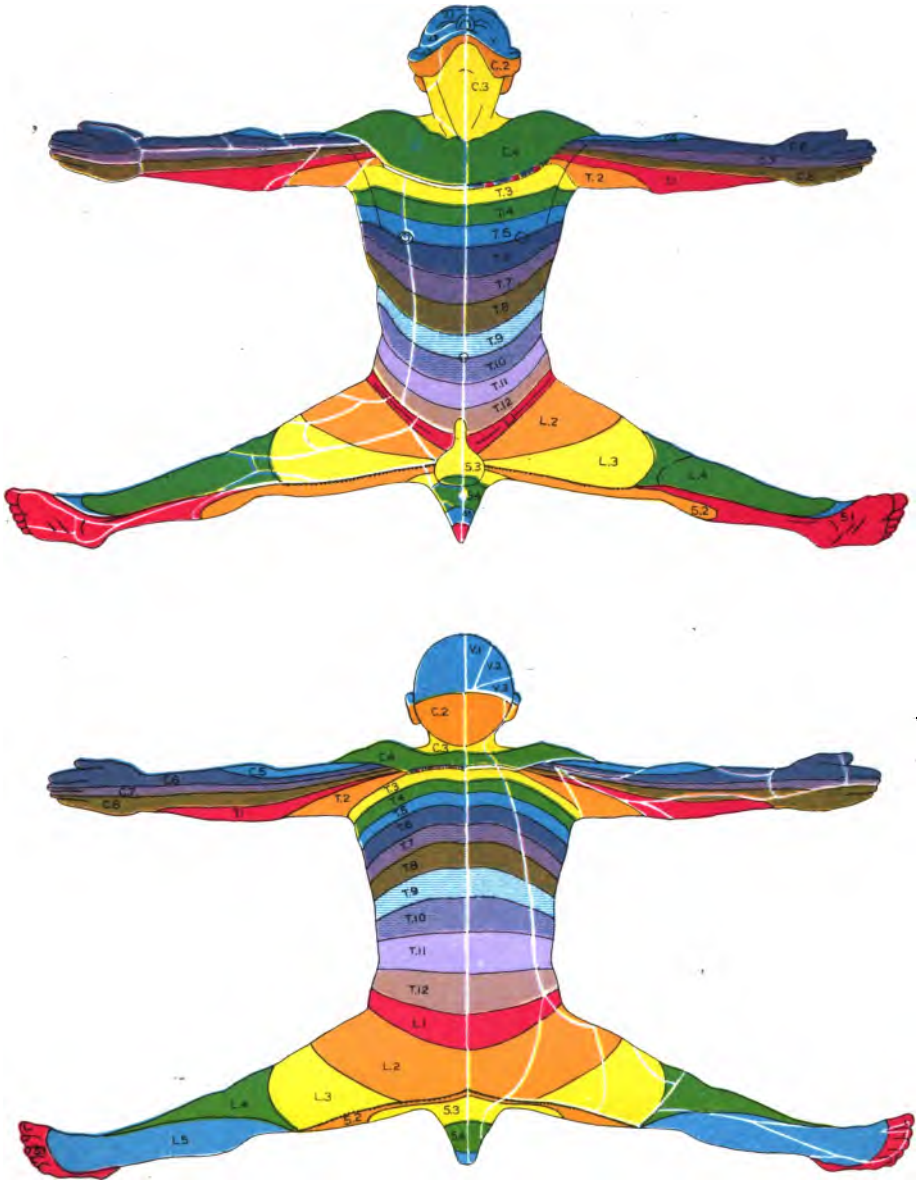

Fig. 126.

Colored Model showing Cutaneous Root-Areas.

Peripheral-Nerve Areas are indicated by white lines on the right half of the model.

scend in the cord in the position indicated. (Fig. 127.) At the various levels fibers pass from this tract to the motor cells of the anterior horn. Through these fibers the anterior horn cells are under control of the higher centers.

II. *Ventral pyramidal tract* (*direct pyramidal tract*).—This tract, as far as is known, subserves the same function as that just described. Its fibers have the same origin and course. At the upper limit of the cord, however, they fail to decussate, and, separating from the fibers of the lateral pyramidal tracts, pass down in the ventromedial

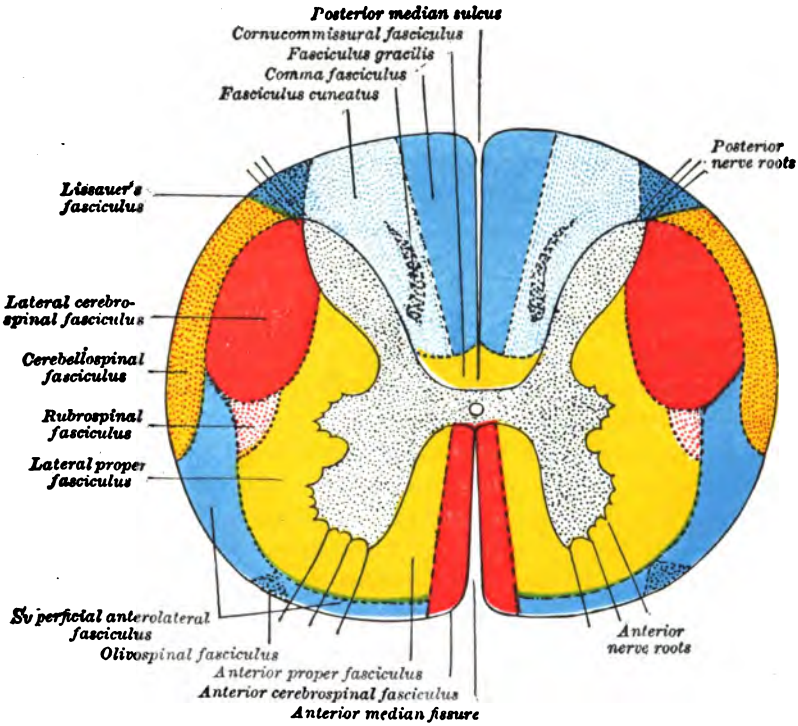

FIG. 127.—Diagram of the principal fasciculi in the medulla spinalis. (Gray.)

aspect of the cord, on either side of the anterior median fissure. Although having the same function (voluntary control of movement) as the lateral pyramidal tracts, they are of much less functional importance.

B. ASCENDING (SENSORY) TRACTS.

I. *Posterior columns* (*columns of Goll and Burdach*).—The posterior column occupies practically the entire dorsal region of the cord, between the posterior horn and the posterior median septum. It receives afferent fibers entering the cord by the posterior roots. It conveys these fibers upward without interruption, c[illegible]me

side on which they enter. It carries them as far as the medulla, where they terminate about the cells of the nucleus gracilis and nucleus cuneatus (nuclei of Goll and Burdach). From here on the tract is continued by a second set of nerve fibers, which decussate in the medulla oblongata, then pass to the optic thalamus of the same side. The posterior columns convey impulses which have to do with the sense of position and passive motion, sense of vibration, discrimination of compass points and touch.

II. *Spino-thalamic tract.*—This tract lies in the antero-lateral region of the cord. Its limits are not as sharply defined as those of the tract just described. The fibers destined for this tract enter the spinal cord by way of the posterior roots, and terminate about the cells of the posterior horn. From here a second neuron carries them forward from the posterior horn, across to the opposite side in the gray commissure, and over to the anterolateral region of the cord. Here they ascend and form the spino-thalamic tract, which continues upward, finally entering the optic thalamus of the same side. It conveys chiefly pain and temperature, and also some touch fibers.

III. *Spino-cerebellar tracts* (*tracts of Flechsig and Gowers*).—These tracts are made up of afferent fibers which enter the cord by way of the posterior roots. They terminate either about the cells of the posterior horns or about the cells of Clark's column. The axons of these cells then pass across to the spino-cerebellar tracts, and upward to the cerebellum by way of the inferior or superior cerebellar peduncles. These tracts are probably concerned with muscle coördination, but their exact function is unknown.

In addition to the tracts just described are certain others, such as the rubro-spinal, vestibulo-spinal, tecto-spinal, spino-tectile, etc., connecting the midbrain, medulla and cord, discussion of which is unnecessary for the present purpose. The rest of the white matter is occupied by short association tracts, connecting the various segments of the cord with each other.

Motor disturbances.—As has been described in a previous chapter, the motor pathway is divided into two parts, the upper motor neuron, and the lower motor neuron. The upper neuron begins with the Betz cells in the pre-central gyrus, and extends down into the cord to terminate about the cells of the anterior horn. Most of these fibers lie in the lateral or crossed pyramidal tract. The lower motor neuron begins with the large motor cells of the anterior horn, and passes by way of anterior roots and peripheral nerve to the muscles.

In the cord a lower motor neuron lesion is produced by involvement of the large motor cells of the anterior horn; an upper motor neuron lesion is produced by involvement of the pyramidal tracts.

Symptoms of involvement of anterior horn cells (*lower motor neuron*).—(1) Paralysis of muscles; (2) atrophy of muscles; (3)

loss of muscle tone; (4) loss of reflexes; (5) reaction of degeneration; (6) fibrillary tremors.

These changes occur only in the muscles supplied by the nerve cells in the particular segment or segments destroyed. They represent a segmental or level lesion. The muscles supplied by the segments above or below the lesion are unaffected. Similarly, any reflex, whose efferent limb lies in the affected area, is lost. Loss of muscle tone and loss of reflexes, are the result of the break in the spinal reflex arc.

The reaction of degeneration comes on gradually in the course of 10 days or two weeks. It will be described in the chapter on Peripheral Nerves.

Fibrillary tremors may occur after any destruction of the anterior horn cells, but are most commonly found in slowly progressive lesions, as seen, for example, in progressive spinal muscular atrophy and syringomyelia.

Destruction of the anterior root fibers, the axons of the anterior horn cells, will produce the same symptoms as destruction of the cells themselves.

Symptoms of involvment of the crossed phyramidal tracts (upper motor neuron lesion).—(1) Weakness of the affected parts; (2) Increased muscle tone—hypertonicity and spasticity; (3) increased reflexes—patellar and ankle clonus; (4) Babinski sign; (5) absence of atrophy; (6) normal electrical reactions.

Lower motor neuron lesions cause paralysis of a muscle or group of muscles; upper motor neuron lesions produce weakness of certain movements rather than paralysis of individual muscles.

Lower motor neuron lesions cause segmental or level disturbances; upper motor neuron lesions cause remote or distant disturbances; that is, all motor functions below the level of the lesion are or may be affected.

The reason for the exaggeration of reflexes and increased muscle tone in pyramidal tract lesions is not clearly understood. The usual explanation is that muscle tone is determined by the spinal reflex arc—a continual stream of tonic impulses passing in through the afferent limb, and out by the efferent limb of the arc. Over this continuous stream of impulses the higher centers have a controlling and inhibiting influence. Removal of that influence by a break in the conduction pathway leads to increased tone, increased reflexes, etc. Patellar clonus and ankle clonus, are an exaggerated expression of this increased tone.

Apparently similar in origin to the increased reflexes and clonus is the phenomenon described by Babinski as the "reflex of defense." In a normal individual, stimulation of the foot often causes some

withdrawal movement. These movements are partly automatic, partly volitional. After division of the pyramidal tract, stimulation of the foot often causes similar but very intense automatic withdrawal movements—dorsal flexion of ankle, flexion of knee and thigh. According to Babinski, these defensive movements may have localizing value; for in cases of marked pyramidal tract lesion it may be possible to elicit them from any area below the level of the lesion but not above the lesion; that is, the highest zone, stimulation of which will produce the reflex of defense, represents the lowermost level of the lesion.

The symptoms above described result from involvement of the pyramidal tract. There is, however, an exception to the rule. Complete severance of the cord at any level, a complete transverse lesion, although destroying both pyramidal tracts, does not produce bilateral symptoms of an upper motor neuron lesion. On the contrary, it results in a complete flaccid paralysis of all the muscles below the level of the lesion, with loss of muscle tone, and disappearance of all reflexes.

Sensory disturbances.—Every posterior root conveys the sensory impulses from a given segment of the body to the spinal cord. Section of that root will cause a failure of these impulses. In general, loss of a single root will cause no perceptible sensory disturbance, owing to the fact that the roots immediately above and below overlap to a sufficient degree to mask the disturbance. When two or three posterior roots are cut there will be a definite loss of sensation corresponding to the region of the body innervated, and involving all modalities of sensation—pain, touch, temperature, deep sensibility. The skin area supplied by each segment is represented in figure 126.

All modalities of sensation are involved following injury to a posterior root, but they are not equally involved. As will be discussed in the section on peripheral nerves, we distinguish, following the work of Head and his collaborators, epicritic, protopathic, and deep sensibility. Epicritic sensibility has to do with light touch and minor degrees of temperature; protopathic sensibility, with cutaneous pain (pin prick), and major degrees of temperature. Deep sensibility has to do with the sense of movement and position, pressure (deep touch) and pain on pressure.

In lesions of peripheral nerves the loss of epicritic sensibility is usually greater than the loss of protopathic. In lesions of the posterior roots, the loss of protopathic sensibility is usually greater than the loss of epicritic. In lesions involving the cord itself all distinctions between epicritic and protopathic sensibility disappears. There ceases to be any distinction between light touch (epicritic) and deep touch or pressure (deep sensibility); between minor degrees of tem-

perature (epicritic) and major degrees of temperature (protopathic); between cutaneous pain (protopathic) and pain on pressure (deep sensibility). Heat, cold, touch, pain—each is lost as a whole or preserved as a whole. From his experience in the present war Holmes has been able to confirm in large measure Head's views.

Irritation of a posterior root will cause pains and paresthesias of radicular type, that is, pains and paresthesias (often associated with hyperesthesia) referred to the particular skin segment which receives its sensory supply from that root.

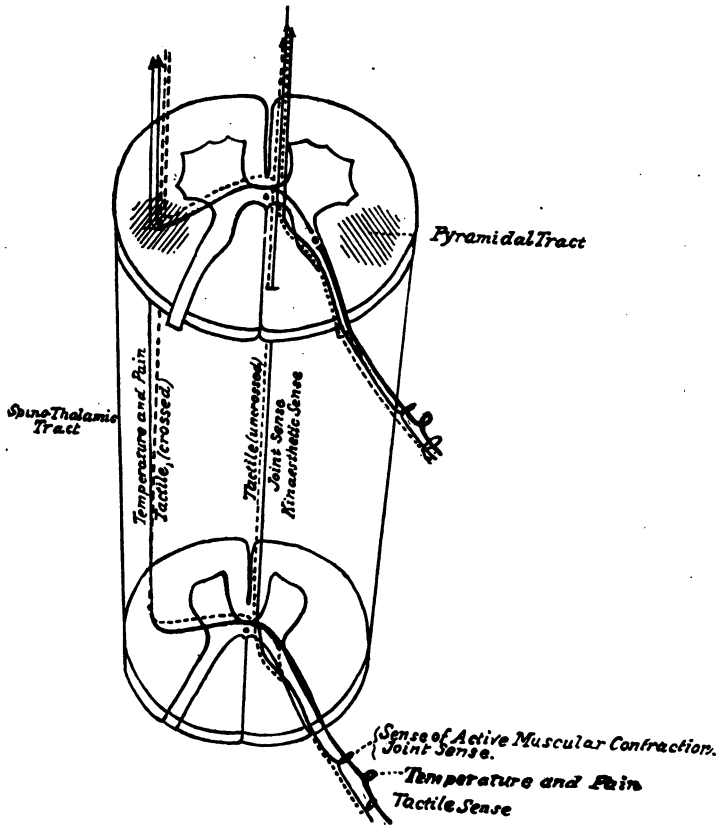

FIG. 128.—Diagram illustrating the course of the various sensory paths in the spinal cord. (Stewart.)

All sensory disturbances produced by localized lesions in the spinal cord are not identical; they depend on the tracts involved, and the modalities of sensation conveyed by these tracts. The course of the fibers representing the different modalities is briefly as follows:

Pain.—The fibers enter the posterior horns, cross to the contralateral side of the cord, and ascend in the contralateral spino-thalamic tract. (Fig. 129.)

Temperature.—The fibers are closely associated with those of pain and have a similar course.

The fibers of pain and temperature do not all cross at the level at which they enter the cord. Many of them ascend one, two, or more segments before passing to the opposite side. From his material in the present war, Holmes believes that the higher the level in the cord, the greater the obliquity of these decussating fibers, and that the obliquity varies from one segment in the mid-dorsal region, to two or three in the upper dorsal, and four, five, or even six segments in the uppermost cervical region.

Touch.—The course of the touch fibers is not positively known. Apparently two pathways are open to them. Some join the pain and temperature fibers, pass to the contralateral spino-thalamic tract, and ascend in that tract. Others enter the homolateral dorsal columns of the cord (Goll and Burdach), and ascend in those columns. In this way the touch fibers ascend in two widely separated tracts, either of which may be destroyed, without seriously interfering with the tactile sense.

Position and passive motion (muscle sense).—These fibers pass directly from the entering root zone into the homolateral dorsal column (Burdach), and ascend in this column. As they ascend they enter the column of Goll and gradually approach the mid-line, being forced inward by overlaying with the successive entering roots. Thus the fibers which enter lowest (lumbar region) come to lie closest to the mid-line.

Vibratory sense.—These fibers have the same course as the muscle sense fibers.

Sensory symptoms following focal cord lesions.—With these facts in view, we can consider what sensory symptoms will result from localized lesions in various regions of the cord cross section. (Fig. 128.)

Lesion at A.—A lesion at this point will involve the entering root zone, catching the fibers of the posterior root just as they enter the cord. It will produce symptoms identical with those of a posterior root lesion, that is, a segmental loss of all sense modalities at the level involved.

Lesion at B.—A lesion at this level will produce a dissociation of sensation. The pain and temperature fibers will be destroyed in their path through the posterior horn on their way to the contralateral spino-thalamic tract. Some touch fibers (4) will also be affected, but not enough to produce symptoms, as other touch fibers (2) from the same posterior root pass up by the homolateral dorsal column and will not be affected by the lesion.

Lesion at B, then, will produce a dissociation of sensation—a loss of pain and temperature with preservation of touch. The sensory loss will be homolateral; it will be a segmental or level loss, only those skin areas being affected whose segments are directly destroyed.

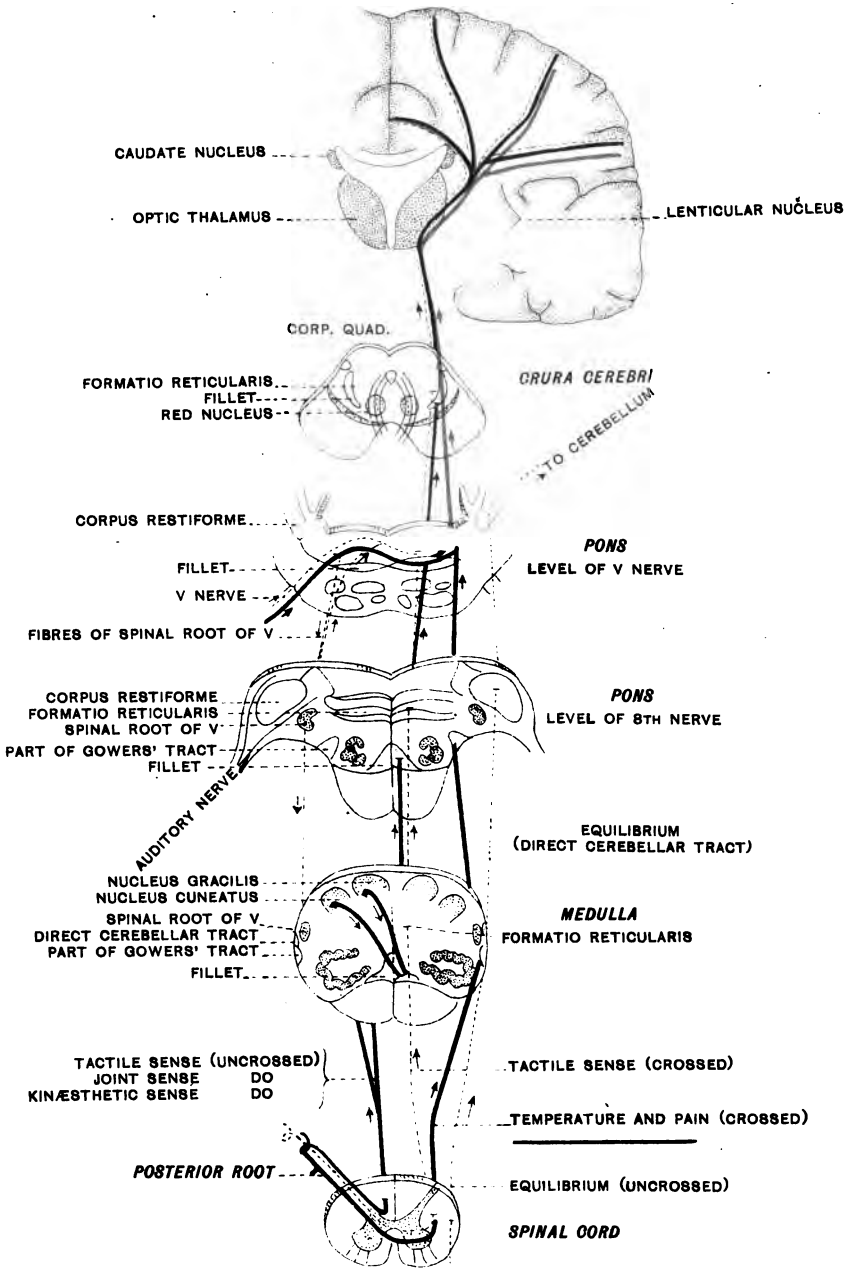

Fig. 129

Diagram of Chief Sensory Tracts in Spinal Cord, Medulla, Pons, and Cerebrum.

(*Fig. 130.*) Lesions in this location are found especially in cases of syringomyelia.

Lesion at C.—Here again we will have a dissociated sensory disturbance—a loss of pain and temperature with preservation of touch. It will be different in two respects from the lesion at B.

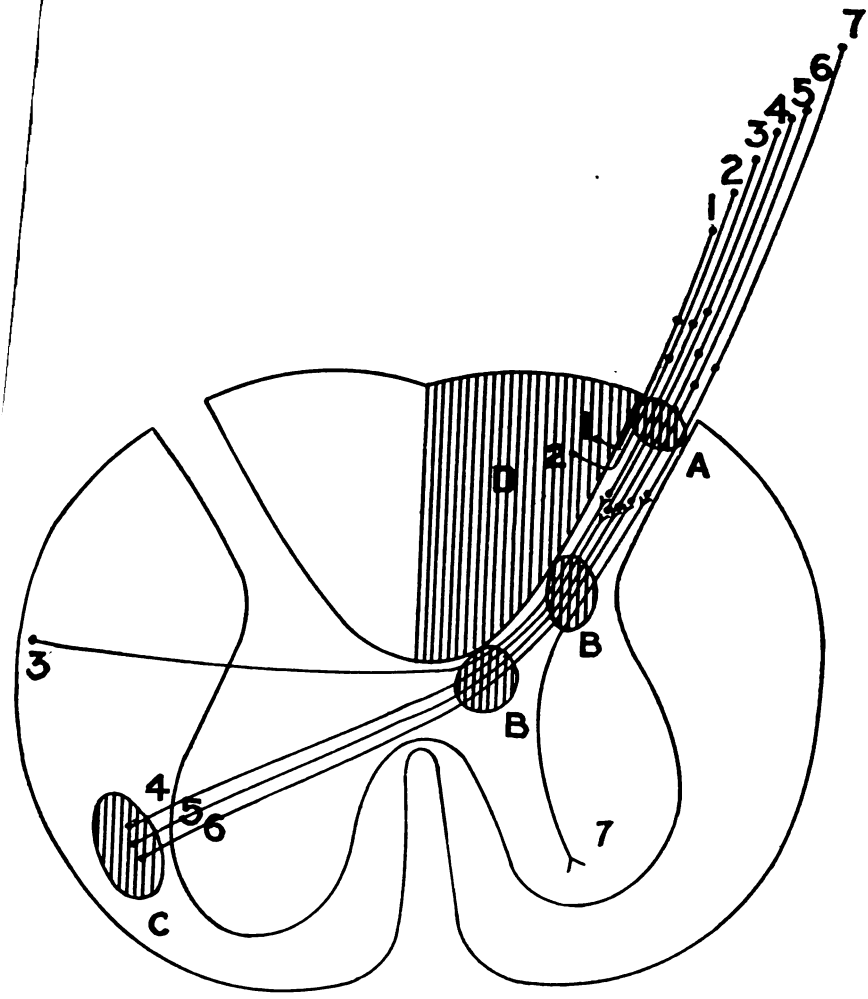

FIG. 130.—Afferent fibers of spinal cord.
1. Muscle sense. 2. Touch. 3. Spinocerebellar. 4. Touch. 5. Pain. 6. Temperature. 7. Fiber to ant. horn cell—afferent limb of reflex arc. (Selling.)

(1) The sensory loss will be contralateral; it will be on the side of the body opposite the side of the lesion.

(2) It will be a "distant" loss, not a level loss. All pain and temperature fibers from the contralateral half of the body below the level of the lesion will be destroyed; for all the pain and temperature fibers which have already decussated at a lower level have come to

lie in the spino-thalamic tract which is here destroyed. This difference in the two types of lesion can be seen in Fig. 131, B. and C.

Lesion at D.—Destruction of the posterior column will cause a homolateral loss of the sense of position and passive motion with resulting ataxia. Touch will be unaffected (because other touch fibers from the same skin areas go by way of the spino-thalamic tract) or

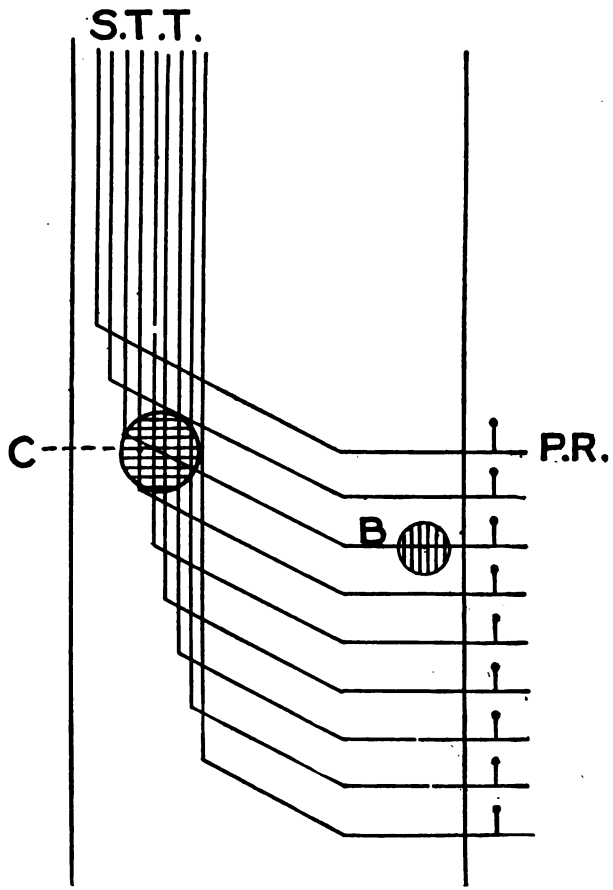

FIG. 131.—P. R., Posterior root. S. T. T., Spino-thalamic tract. Showing the pain and temperature fibers in their course from the posterior root across and up the spinothalamic tract. (Selling.)

only moderately affected. Sense of vibration will be lost on the same side. Bilateral lesions of this type occur especially in tabes dorsalis.

As in the case of destruction of the spino-thalamic tract, so in the case of the posterior column destruction, we are dealing, not with a level symptom but with a distant symptom. All the muscle sense fibers collected from all the posterior roots at lower levels have entered the posterior column, and are destroyed by the lesion. Section of one posterior column in the thoracic region, for example, will

cause homolateral loss of sense of position and passive motion in ankle, knee, and hip.

Bladder and rectum.—Disturbance of bladder and rectal functions is a frequent concomitant of disease of the spinal cord. The centers for these organs lie in the gray matter of the conus terminalis, especially the third and fourth sacral segments. Destruction of these centers, or the corresponding anterior roots, causes complete incontinence.

Destruction of the afferent roots causes disturbance of function through loss of sensation (as in tabes dorsalis). Finally, pyramidal tract lesion at any level, especially bilateral pyramidal tract lesion, produces disturbance of varying grade. Voluntary bladder control is lost, and the bladder may empty itself spontaneously, at intervals, or become overdistended, and overflow.

The centers for sexual function are closely associated with centers for bladder and rectum, and are frequently affected with these.

Localization of lesions in the cord.—The various tracts of the spinal cord have been briefly described. The symptomatology of disease or injury of the cord resolves itself into a grouping of symptoms which result from involvement of these various tracts; in each case it will depend upon the nature, extent, and localization of the pathological process.

Two general types of symptoms must be distinguished—level symptoms, and distant symptoms. This holds for both motor and sensory phenomena. Taking by way of illustration a destructive lesion involving one-half of the cord at the level of the eighth cervical and first dorsal segments the following symptoms will result:

1. Homolateral weakness of the small hand muscles, of the lower motor neuron type. This is due to destruction of the anterior horn cells which innervate these muscles and which lie in the eighth cervical and first dorsal segments. It is a level lesion.

2. Homolateral weakness of the leg of the upper motor neuron type, with spasticity and increased reflexes. It is due to destruction of the pyramidal tract. It is a distant lesion, i. e., affects all the parts below the level of the lesion.

3. Homolateral anesthesia in the eighth cervical and first dorsal region—that is, the under half of the arm. It is a complete anesthesia, affecting all modalities of sensation, and is due to destruction of the two posterior roots as they enter the cord. It is a level symptom.

4. Homolateral loss of muscle sense due to section of the posterior column (Goll and Burdach). This is a distant symptom, affecting all joints and muscles below the level of the lesion.

5. Contralateral loss of pain and temperature with preservation of touch (dissociated sensory disturbance), due to destruction of the spino-thalamic tract. It is a distant symptom, affecting

half of the other side of the body below the level of the lesion—chest, trunk, and leg.

The symptomatology of lesions involving a greater or smaller portion of the cross section of the cord can be derived from the above. It will vary considerably, depending on whether the involvement is uniform or, as in many cases of myelitis, more or less patchy. The pyramidal tract symptoms with weakness of the legs, spasticity, increased reflexes, clonus and Babinski, are usually prominent in all lesions above the lumbar enlargement. If the lesion is high enough (upper cervical cord) the arms will also show symptoms of pyramidal tract involvement.

The diagnosis of the level at which the cord is diseased or injured will depend upon motor, sensory, and reflex disturbances which characterize the different levels. Fig. 126 gives the skin area supplied by each segment of the cord. Table V gives the segments in which lie the anterior horn cells for each of the more important muscles. And Table VI gives the segments of the cord which hold the reflex arcs for the various skin and tendon reflexes. The following contains some of the more characteristic types of disturbance.

Cervical cord.—C1–C5. Injuries in this region are often rapidly fatal, due to the proximity of the important vasomotor and respiratory centers in the medulla. The diaphragm, which is supplied from C3–4 may be paralyzed on one or both sides. Hyperthermia is common.

C5 and C6. Involvement of these segments as of the corresponding roots will affect the same group of muscles as are involved in cases of obstetrical palsy (Erb-Duchenne type)—deltoid, biceps, brachialis anticus, and supinator longus. The biceps reflex will be lost.

C7. Destruction of this segment will cause weakness or paralysis of the triceps and loss of the triceps reflex.

C8 and D1. Lesions at this level as of the corresponding roots will affect the same group of muscles as are involved in the so-called lower arm type of lesion or Dejerine-Klumpke type. The characteristic feature is paralysis and atrophy of the small hand muscles. The white rami passing to the cervical sympathetic leave the cord at this level, and destruction of these fibers causes contraction of the pupil, narrowing of the lid slit, and enophthalmos on the same side (see Cervical Sympathetic).

Dorsal cord.—In determining the level of lesions in the dorsal region, examination of the intercostals and the abdominal muscles is of value. If the fingers are placed in the intercostal spaces, the unaffected muscles are felt contracting on each deep inspiration, while in the paralyzed spaces no contraction can be felt and the

finger sinks in between the ribs. As each intercostal muscle receives its nerve supply from the correspondingly numbered dorsal root, the highest space which is paralyzed indicates the level of the spinal injury.

The abdominal muscles are tested by coughing, sitting up, or raising the head. If the ninth dorsal is involved, the lower portion of the rectus from about an inch above the umbilicus is paralyzed, but the upper part intact. On contracting the abdominal muscles, the umbilicus is drawn upward toward the xiphoid (Beevor's sign). If the eleventh dorsal is involved, the whole rectus is intact, but the iliac regions bulge, owing to paralysis of the lower portion of the abdominal oblique.

Lesion of D8–9 will cause disappearance of the upper abdominal reflex.

Lesion of D10–2 will cause loss of the lower abdominal reflex.

Lumbar and sacral cord.—Lesions of L1–2 cause loss of the cremasteric reflex.

Lesions of L2–3–4 cause loss of the patellar reflex, paralysis of the quadriceps extensor and perhaps also of the tibialis anticus.

Lesion of L5, 1S (epiconus) cause disappearance of flexor response on stimulating the sole of the foot, loss of the Achilles reflex, and paralysis of the muscles of the antero-external surface of the leg.

Lesions of S3–4 cause loss of bladder and rectal control, with incontinence of urine and feces.

In determining the level of cord lesions by means of sensory disturbance, we must recall the difference between lesions of the spinal roots (or entering root zone) and of the spino-thalamic tract.

Where the roots, or entering root zone are affected, touch, pain, and temperature are all involved, and the *level of the sensory disturbance corresponds to the level of the lesion.* Where the spino-thalamic tract is affected, the sensory loss is of the dissociated type, and the level of the *sensory disturbance is one, two, or three segments below the level of the lesion.* This is due to the fact that one or more segments are required before the pain and temperature fibers, passing from the posterior root to the contralateral spino-thalamic tract, have completely decussated.

From the description given above, it will be possible to deduce the symptomatology of spinal cord lesions of varying extent at the different levels. Certain special cases may be briefly discussed.

Complete transverse lesion.—In partial injuries of the cord, the involvement of the pyramidal tracts causes weakness, spasticity, increased reflexes, clonus and Babinski. On the sensory side the symptoms are commonly of the dissociated type.

In complete transverse lesions there is complete loss of motion and of all forms of sensation below the level of the injury. The paralysis

is of the flaccid type, with atony of the muscles and disappearance of all reflexes both deep and superficial. Stroking the sole of the foot produces no response (neither flexion nor extension of the toe). There is no reflex of defense. There is complete loss of bladder and rectal control, and loss of sexual function.

Brown-Séquard syndrome.—The Brown-Séquard syndrome results from unilateral lesion (hemisection) of the cord. It has already been described, and a brief resumé will suffice.

Homolateral symptoms: (*a*) Weakness with increased reflexes, spasticity, etc., due to interference with the pyramidal tract. (*b*) Disturbance of muscle sense with ataxia, due to involvement of the posterior column.

Contralateral symptoms: (*c*) Loss of pain and temperature sense.

Vasomotor symptoms may occur on the side of the lesion, cyanosis, and diminished temperature of the part.

Hyperesthesia of the skin on the side of the lesion and at the level of the upper limit of the lesion is often found. It results from irritation of the entering root fibers.

Hematomyelia.—Hemorrhage into the substances of the cord as the result of trauma is not uncommon. It may accompany a fracture or dislocation of the vertebra, or occur independently of these. The seat of the hemorrhage is usually in the gray matter, and it may extend for a considerable distance up and down the cord in the central gray. The white matter is much less frequently involved.

The symptoms of hematomyelia are determined by this localization. The pain and temperature fibers are cut as they decussate. There is, therefore, pain and temperature loss at the level of the hemorrhage. The spino-thalamic tract itself is not affected, so there is no sensory loss below the level of the lesion. By extending into the anterior horn, the anterior horn cells will be destroyed, and a flaccid paralysis result at the level.

The essential symptoms of hematomyelia are, then, dissociated sensory disturbance and flaccid paralysis, corresponding to the level of the hemorrhage.

Small scattered hemorrhages may occur in the cord, without causing any recognizable symptoms.

Hemorrhage may occur within the dural sac. Unless this is very extensive, it is not apt to cause immediate symptoms. Symptoms may develop later on, as a result of gravitation of the blood to the lower end of the sac, clotting, and organization of the clot. This may produce pressure symptoms and especially pain, by compression of the roots of the cauda equina.

Fracture and dislocation of the vertebræ.—Dislocation of the vertebræ is most common between the fifth and seventh cervical spines and

between atlas and axis. Fractures are most common in the mid-cervical and upper dorsal regions, and especially from the tenth dorsal to the first lumbar spine. The two conditions are often associated. Objectively a deformity is usually recognizable at the site of the lesion, along with the rigidity of the spine and pain in the back.

In some cases where there has been sufficient trauma to cause a fracture or dislocation of the vertebræ, the cord itself may be severely damaged. The symptoms of fracture and dislocation of the vertebræ depend mainly on the degree of damage to the cord and the nerve roots. The cord may be crushed by the injury or compressed by in-driven fragments of bone. Sometimes there is a temporary dislocation of the spine, the dislocated vertebræ springing back into place immediately following the cessation of the trauma. In that instant of dislocation, however, the cord may be completely crushed.

The degree of the damage will vary greatly in different cases; the symptoms will depend upon the degree of damage, and the level at which it takes place. This has already been discussed in dealing with the symptomatology of the cord lesions at varying levels.

Myelitis.—The concept of myelitis is not a very clearly defined one. It is usually held to include not only inflammatory conditions, but also diffuse or scattered foci of softening such as are apt to accompany trauma, often affecting parts of the cord more or less remote from the seat of injury. These are discussed in some detail in the article by Holmes.

The inflammatory types are infectious or toxic in origin. Any general infection may give rise to infection within the substance of the cord; and any general intoxication may give rise to a toxic myelitis.

The myelitic process may be disseminated or focal. In the former case, small foci are found scattered more or less widely throughout the length of the cord. In the latter case, it may involve a part or the whole of the cross section of the cord at any level, and give rise to symptoms varying from the mildest, to those of complete transverse lesion.

Cauda equina.—The spinal cord terminates at the level of the lower border of the first lumbar spine. (The lowermost part of the cord including the third, fourth, and fifth sacral segments is called the conus medullaris). Below the end of the cord are grouped together the lumbar, sacral, and coccygeal roots to form the cauda equina. These roots arise close together in the cord and run down a variable distance before passing out at their respective levels. The anterior and posterior roots arising from each segment of the cord remain

separate throughout the entire intra-dural course. The posterior root ganglia lie just outside the dura.

Symptoms of lesion in this region will vary in extent according to the particular roots injured and the extent of the injury to each.

Lesions of the cauda are lesions of nerve roots and the motor and sensory loss following such lesions will be distributed according to root or segmental type and not according to peripheral nerve type.

Symptoms.—Complete destruction of the cauda produces complete paralysis of the lower limbs, anesthesia to just about Poupart's ligament in front, and the upper sacrum behind, including the perineum and genitalia; loss of bladder and rectal control and of sexual functions; loss of knee jerks, Achilles reflexes and plantar response.

In lesions about the level of the first lumbar vertebra, the conus medullaris, consisting of the third, fourth, and fifth sacral segments of the cord, may be destroyed. Injuries in this region may involve conus, or cauda, or both. The diagnosis between the two is often difficult. Isolated destruction of the conus medullaris will result in loss of function of the bladder and rectum, loss of the anal reflex, and anesthesia in the region supplied by the third, fourth, and fifth sacral roots; that is, a saddle-shaped area round the anus. Where there is a combined lesion of conus and cauda, the caudal symptoms usually mask those of the conus, and differential diagnosis is impossible. Dissociation of sensation points to involvement of the conus. Asymmetry of the sensory phenomena, points to a caudal lesion.

The conus ends at the first lumbar vertebra, so that an injury below this level can involve only the cauda; the symptoms will vary according to the level of the lesion and to the extent of the involvement.

Complete destruction of the cauda at the level of the first lumbar vertebra, will cause complete paralysis of the legs.

Below the level of the second lumbar vertebra an injury will spare the iliopsoas, and some flexion of the thigh will be possible.

Below the level of the third lumbar vertebra an injury will spare the quadriceps femoris. Extension of the leg will be possible and the knee jerks will be preserved. The hamstring muscles and all the muscles below the knee will still be paralyzed. There will be loss of Achilles reflexes and of bladder and rectal control.

Below the level of the second sacral vertebra there will be no paralysis of the leg muscles, as these are all supplied from higher roots. Knee jerks and Achilles reflexes will be retained. There will be a saddle-shaped area of anesthesia over the buttocks, including perineum and genitalia.

Below the third sacral vertebra an injury will spare the bladder and rectum. There will be paralysis of the perineal muscles, and anesthesia immediately about the anus.

In the cauda, motor and sensory roots can be separately injured.

TABLE VII.—*Cauda equina.*

	Sensory distribution.	Motor distribution.
First and second lumbar	Groin, upper part of thigh, and buttock	Psoas, iliacus, and pectineus.
Third and fourth lumbar	Anterior, inner and outer surfaces of thigh, the knee, and inner side of leg.	Quadriceps extensor cruris, adductors, and tibialis anticus.
Fifth lumbar	Antero-external surface of leg and dorsum of foot.	Extensors of toes and peronei.
First sacral	Sole of foot and calf	Muscles of sole and calf.
Second sacral	Popliteal space, posterior surface of thigh and lower edge of buttock.	Hamstrings and glutei.
Third, fourth, fifth sacral and coccygeal.	Perineum and genitals	Levator ani and perineal muscles.

War injuries.—The following account of the spinal injuries of warfare is taken from the articles by Gordon Holmes[1] and James Collier.[2]

The spinal cord may be injured directly by a projectile and either completely or incompletely divided; more often it may escape direct damage by the missile, and be compressed or lacerated by displaced fragments of bone. Frequently, it may escape injury from either of these sources, and yet marked structural changes may be found. This occurs when the projectile has struck a spinous or transverse process or has penetrated the body of a vertebra, the so-called impact lesions.

A. Direct injury.—Where the cord has been completely divided blood clot and pieces of bone are usually found between the severed ends. A few days after the infliction of the wound these ends are swollen, irregular, and very soft to the touch for at least 1 cm. from the point of division. They may even be more or less diffluent. The outlines of the cross section are obscured, and smaller or larger hemorrhages are often seen, chiefly in the gray matter.

Secondary changes.—For at least half a segment above and below the actual site of the lesion secondary changes occur. They are degenerative rather than inflammatory in type. They consist of edema, circulatory disturbances, and the formation of peculiar cyst-like cavities containing necrotic material, detritus and fluid under pressure. These cavities are connected with the site of the lesion by means of a small canal, or slit, or by a track of softened cord substance. They are most common in the tip of the posterior columns. All these changes are capable of producing secondary destruction of the cord tissue.

Distant lesions.—Edema of both gray and white matter, with some swelling and softening of the cord, is the most constant of these changes. They diminish gradually away from the wound and often seem to bear no relation to its severity. There may be vacuolation

[1] Spinal injuries of warfare. *Brit. Med. Jour.*, Vol. II, 1915.
[2] Gunshot Wounds and Injuries of the Spinal Cord. *Lancet*, 1916, I, p. 711.

due to falling out of fibers, and a sieve-like rarefaction produced by the disappearance of fibers of the finer glial matrix.

Hemorrhages of various sizes are often associated with the edema; they are generally small and punctiform, giving a mottled appearance to the cross section. They are found particularly in the gray matter about the central canal and in the dorsal horns, and may extend for a considerable distance up and down the cord. They cause relatively little tissue destruction.

B. Contusion or compression of the spinal cord.—When a portion of a vertebra or a detached spicule of bone is driven into the spinal canal it may lacerate both cord and dura and cause lesions which differ only in degree from those produced by the projectile. Frequently, however, without any sign of external injury to the cord and with dura intact except for small hemorrhages there may still be a complete crush. On palpation the cord at this level is soft, and on incising the pia semifluid cord substance escapes.

Where the damage to the cord is less severe complete softening of the injured area does not occur. The normal appearance of the cross section is obscured but not wholly lost. There are irregular foci of softening especially in the ventrolateral and dorsal columns. There is some disintegration of the gray matter with cell necrosis and chromatolysis. Edema and hemorrhages are present, and after a time there is invasion by granule cells and proliferation of pia. Distant changes are proportionately slight.

C. Impact lesions.—Where a projectile strikes a spinous process or transverse process severe damage of the cord substance may result from the force of the blow, although the cord has not been touched by the projectile nor by bony fragments. These are called impact lesions. Macroscopically no abnormality may be recognizable, or at most there may be a more or less uniform swelling opposite the site of the blow. Microscopically there are edema and diffuse or focal necrosis, most marked in that part of the cross section nearest the point of impact. In these areas there may be complete destruction of all functional elements, but usually only a portion of the fibers have disappeared, while others show swelling of their sheaths or axis cylinders. Focal softenings also occur in the gray matter, while punctiform hemorrhages are apt to be abundant here.

The distant lesions in cases of impact lesion are similar to those found associated with direct and contusion injuries, but they are often very marked in relation to the changes found at the site of maximum damage. Scattered hemorrhages, irregular foci of necrosis, softening and cavity formation occur; but the most pronounced feature is the extensive parenchymatous change that often extends over four or five segments in either direction. These consist in the swelling of fibers, in the midst of tissue which is otherwise

normal or only slightly edematous; as a rule, the axis cylinder is more swollen and the myeline sheath surrounds it as a distended and attenuated ring; but in places the sheaths are more affected, and are often broken up. The disappearance of these swollen fibers and the partial disintegration of the neuroglia leave vacuoles and round or oval cavities in the white matter and give it a reticular or sieve-like appearance. There are also considerable histological changes in the nerve cells at some distance from the level of the injury.

The mechanism of production of these anatomical changes in the cord following an impact lesion, without direct injury to the cord itself, is not clearly understood. It is probable that some of the distant lesions described in cases of direct injury, which are beyond the limits of the injury itself, are of similar nature.

Secondary changes—It has occasionally happened that when the wound is not severe, the patient was at first able to perform some movements in his legs, but lost the power to do so within the following two or three days. Further new symptoms may develop, or their level alter under observation. This may be due to secondary hemorrhage into the affected tissue, or to progressive softening. But Holmes believes it is often due to the formation of cylindrical cavities—cyst-like cavities somewhat analagous to retention cysts—which are formed from fluid under pressure, tracking up and down the cord from the site of the primary injury.

It is evident that these irrgular and diffuse changes which are found in the various types of spinal injury must be taken into account in interpreting and estimating the significance of the clinical symptoms that are observed in these cases. The following conclusions, at least, can be drawn:

1. The structural lesions in the spinal injuries of warfare are rarely sharply limited or circumscribed, and can not be compared to those produced experimentally in a physiological laboratory. The level of the lesion, as indicated by the clinical symptoms, for instance, often does not correspond with the level of maximal damage.

2. The lesions are so irregular in distribution and severity when the spinal injury is not complete that much care is necessary in drawing conclusions from the clinical symptoms alone on the functions of parts which it may be assumed have been involved.

3. Secondary changes may occur later in the cord which can alter or modify the clinical symptoms.

In addition to the lesions just described certain other types must be considered.

D. Concussion lesions.—Severe indirect injuries of the spinal cord may occur from the bursting of high-explosive shells when the back is turned toward the force of explosion, without any external wound

occurring, without any lesion of the bones, and even without bruising of the soft tissues. Of the cases of this type which Collier reported, the lesions were all in the lower dorsal region.

E. Root lesions.—Sometimes the lesions produced by projectiles affect the spinal roots after they have left the thecal space to a greater extent than they affect the spinal cord and thecal contents. This seems to be the case when there has been much fracture and crushing of the bone, especially in the region of the transverse processes. There is usually much swelling and edema in the injured region of the spine. Such root lesions are not entirely due to direct injury, for they may be widely spread when the injury is comparatively local. They may result from sub-periosteal hemorrhages and periosteal swelling, which strangle the roots in the intervertebral foramina; or from pachymeningeal hemorrhage.

F. Intrathecal hemorrhage.—Blood effused within the dura tends to sink to the bottom of the dural sac. Unless the hemorrhage is very extensive it is not apt to produce many symptoms. Subsequently, as a result of clotting, with organization of the clot and contraction of the fibrous tissue, pressure and destruction of the roots of the cauda equina may result. And this may be sufficiently extensive to produce the physical signs of a complete lesion of the cauda. Partial lesions of a similar nature may occur, and the possibility should always be considered where some of the physical signs (as, for instance, local wasting of muscles, and local loss of faradic excitability) are difficult of explanation.

The diagnosis of this condition at such a time as to allow of the removal of the blood by lumbar puncture is almost impossible; for nearly all the spinal lesions resulting from bullet wounds show symptoms of a complete transverse lesion at first, and therefore the development of symptoms of involvement of the cauda equina can not be detected at this stage.

Reflexes and reflex tone.—Following all severe lesions, the lower limbs become flaccid. This flaccidity develops within 24 hours of the infliction of the wound. If the lesion is complete or almost complete the flaccidity persists and is followed by atrophy. If less severe the flaccidity is followed by a gradual return of muscle tone, and, generally within two or three weeks, the limbs become more or less rigid and spastic.

As for muscle tone, so also for the tendon reflexes. Except where the spinal lesion is slight, knee jerks and ankle jerks are lost at first. With complete transverse lesions, the loss is permanent. With less severe lesions the reflexes return generally after two or three weeks. In general the knee jerk returns before the ankle jerk.

In unilateral lesions of the cord, there is a homolateral flaccidity and loss of tendon reflexes which follow the same general rules as

those which obtain in cases of severe transverse lesions. In such cases, however, the return of reflexes occurs somewhat earlier than in transverse lesions of the same degree of severtiy.

With regard to the superficial reflexes, the abdominal and cremasterics are more easily abolished than the tendon jerks, and when the lesion lies above the mid-dorsal region, remain permanently absent as long as there is any obvious motor weakness of the lower limbs.

Plantar response.—Normally, stimulation of the sole produces a complicated response, consisting of flexion of the great toe, and a "withdrawal movement"—flexion of the knee (hamstrings), flexion of the hip, adduction of the hip, etc. As in the case of muscle tone and deep reflexes, after all severe spinal injuries this response is diminished or lost, and the degree of the diminution depends on the extent of the lesion. Where the lesion is complete or nearly so, stimulation of the sole produces no movement of the toes whatever. Where it is less complete, stimulation produces a simple flexion of the great toe. This movement differs from the normal flexion response in that it is slower and smaller in range, and is called by Collier the "reduced flexor response." In still less severe injuries stimulation of the sole evokes flexion of the great toe plus contraction of the hamstrings; while in the least severe, a complete withdrawal reflex might be obtained, still with flexion of the great toe. In some of these cases extension of the great toe occurs instead of flexion—Babinski's sign. The occurrence of a Babinski within the first week or 10 days means that the lesion is not complete.

This applies to the period following the injury. The subsequent events depend upon the severity of the lesión and degree of recovery. With very severe lesions, the absence of response or reduced flexor response may remain unchanged. In more favorable cases they gradually give place to an extensor response. If complete recovery ensues, the extensor response finally changes back into a normal flexor response.

Spinal shock.—The abolition of the tendon jerks and the absence of Babinski's sign, found in the early stages following severe but not necessarily complete transverse lesion of the cord, is known as "spinal shock." It is the result of a sudden isolation of a portion of the cord from the rest of the central nervous system. This sudden isolation leaves the cord incapable, for a time, at least, of observing even the simplest reflex. After the state of shock has passed off, the reflexes will remain the same, or will change, depending on the degree of the anatomical lesion.

The unilateral absence of reflexes following a uninlateral lesion shows that this abolition is not due to a state of general shock or to

a gross injury of the cord, but that it is due to an interruption of impulses which descend through the homolateral half of the cord. The effects of spinal shock on the responses of the lower limbs is the same whether the lesion is in the dorsal or cervical region.

Special symptoms.—*Cervical enlargement.*—Incomplete lesions. Hemorrhages and secondary changes occur as a result of concussion more readily in the cervical region than in any other region of the cord. Inasmuch as they are more liable to damage the gray than the white matter, the arms are often seriously paralyzed, whereas the legs are relatively little affected. The paralysis may develop some time after the infliction of the wound. In some cases where all four limbs are involved at first, there is rapid return of power, more rapid in the legs than the arms.

Paralysis of the diaphragm, unilateral or bilateral, was found in lesions at the fourth and fifth cervical segments, or somewhat higher. It was never found in lesions below the fifth.

Paralysis of the cervical sympathetic was found in lesions anywhere between the second cervical and second dorsal segments. Miosis, enophthalmos, ptosis, diminished sweating and sometimes reddening of the affected side occurred. Only paralytic symptoms were observed, never symptoms of irritation. The symptoms were identical whether the injury affected the descending sympathetic tracts in the cord, or the spinal sympathetic centers themselves in the eighth cervical, first and second dorsal segments. Where the centers were affected, however, the disturbance seemed more severe, and more persistent.

Lower portion of the cervical enlargement.—Severe lesions in this zone gave rise to a more or less characteristic group of symptoms. These were, (1) subnormal temperature; (2) slow pulse; (3) low blood pressure; (4) scanty secretion of urine. All of the cases in which this symptom complex occurred died within 10 days.

The skin was cyanotic, and cold to the touch. The temperature in one case reached as low as 78.8° F., and in others from 80 to 84° F. The pulse rate in one case was 22 per minute; in others it ranged from 30 to 50 per minute. It always ran parallel to the temperature, rising and falling with it. The blood pressure taken in three cases showed, respectively, 56, 72, and 73 mm. mercury. The urine was greatly reduced, one patient having complete anuria for 48 hours, another secreting only 20 ounces in 3 days. The patients were stuporous and lethargic, but could be aroused. If the temperature rose, the mental state would rapidly clear. Post-mortem examinations of a number of these patients showed the lesion to lie between the sixth cervical and the first dorsal segments. Other cases were seen, however, with severe lesions in these same regions, in which the symptom complex was not present.

Mid-dorsal region.—When the mid-dorsal region is severely injured, the abdomen is frequently tense and distended, and the patient presents symptoms of paralytic distension of the intestines. A striking symptom which is sometimes associated with it, but which often occurs without any objective symptoms of abdominal disturbance, is vomiting. It may begin within a few hours of the infliction of the wound, and be so severe and persistent as to threaten life. It is often projectile. Most of these patients complained of girdle pains, and paresthesias between xiphoid and umbilicus. These phenomena are probably the result of irritation or injury of the sympathetic fibers to the stomach. At times they suggested the vomiting of tabetic crises.

Sensory disturbances.—According to Holmes, a lesion can be localized as accurately from the motor paralysis as from the upper border of the sensory disturbance. In incomplete lesions, and especially in those which are wholly or chiefly unilateral, an exact local diagnosis may not always be possible from even an accurate sensory chart, due to the oblique course of the decussating sensory fibers in the cord.

Anesthesia to pain and temperature is the most common and prominent disturbance of cutaneous sensibility. Both are lost on the side opposite the lesion when this is unilateral, and more diminished on the side opposite the greater injury when this is bilateral. This crossed relation maintains only when the lesion is above the first lumbar segment. Below this, the pain and temperature fibers are caught before they have a chance to decussate. The contralateral loss of pain and temperature above described is, of course, a distant lesion. Remote (distant) tactile anesthesia is rare in unilateral lesions. When it occurs it is usually on the same side as the loss of pain and temperature. Where tactile sensibility is involved as a result of bilateral lesions, along with pain and temperature, it usually recovers before the latter.

When the lesion is not complete, and especially if it is unilateral, the upper border of the sensory loss frequently retreats caudalwards in the course of time. Further, where the lesion is high in the cord, and has produced marked or total loss of sensation to the corresponding level, it is not uncommon to find the area of the lower spinal roots, and especially the skin in the region of the anus, sensitive to one or other mode of stimulation; or, in case of complete sensory loss from a high lesion, sensation may begin to return first in these sacral root zones. These phenomena indicate that there is a lamellar arrangement in which the fibers that carry any specific form of sensation from successive dorsal roots lie in series.

Subjective sensory symptoms.—*Distant pain*, in the leg, for instance, when the spinal wound was in the cervical region, occurred in

some cases. Usually it was transient, but sometimes persistent. It occurs only with relatively slight or unilateral lesions, and in the latter case was always on the same side as the injury. *Homolateral hyperesthesia* occurred in some of the Brown-Séquard cases, extending over the whole half of the body, almost as high as the segmental level of the injury. *Finally hyperesthesia at the upper level of the sensory loss* on one or both sides, was frequently found with either complete or partial lesions. It is usually attributed to irritation of the posterior roots, but Holmes believes that in some cases, at least, it is due to edema or circulatory changes at the corresponding cord levels.

Prognosis.—Although neither cells nor fibers of the spinal cord regenerate, very considerable improvement may occur, as at least part of the early symptoms are due to edema and circulatory changes.

The prognosis during the first two weeks is extremely difficult. In nearly all cases the initial clinical picture is that of a total cord lesion, and there is no positive way of telling how much of the transverse lesion is anatomical and how much physiological.

The reflexes give us the best information. Early reappearance of the plantar reflex, if lost at first, and the change from the flexor to the extensor type soon after the seventh day, or the presence of an extensor response earlier than this, are certain indications that the lesion is partial and that some recovery will occur. Persistent loss of the plantar reflex, and persistent flexor response, are indications that the lesion is severe and that useful recovery is highly improbable. Early return of the knee jerk and ankle jerk is of good prognostic import. Even in less severe cases the knee and ankle jerks do not return before the fourth day, and their reappearance before this date gives hope that complete recovery may occur.

Further prognostic indications depend on the date of return, and the rapidity of return of sphincter control, of sensibility and of motor power. Early return of sphincter control is one of the best signs.

A large proportion of cases of spinal injury die soon after the infliction of the wound from shock, or associated wounds of the chest or abdomen. Among those that survive, the greatest danger is from cystitis and pyelonephritis and the development of extensive bed sores. Even with the greatest of care in catheterization, bladder infections are almost certain to occur. In some cases suprapubic drainage is of value.

Decubitus ulcers are preventable in the majority of cases by careful nursing. Avoidance of soiling with urine and feces, the use of air cushions, and frequent turning of the patient to avoid prolonged compression will all help to prevent the development of this most serious of complications.

The Röntgenographic examination of the spine.—In discussing injuries of the spine we have to deal with a very complex system of bones and joints. In addition to the usual effects of trauma to osseous and articular structures there must be considered the serious results of injury to the spinal cord which the vertebræ inclose and protect. In most regions of the body, fractures and dislocations are discussed separately, but in the spine the two injuries are so frequently associated, and uncomplicated dislocations are comparatively so infrequent, except in the cervical region, that the two conditions are usually considered together as fracture-dislocations. This term implies a fracture together with an associated permanent disjunction with displacement, which can be shown by the röntgenogram or found at operation. It should be borne in mind, however, that a fracture may be accompanied by a transitory dislocation, which becomes immediately replaced, but which may have been in reality the factor concerned in a cord injury. In such injuries the only evidence found by a röntgen examination may be a slight fissure through the body of a vertebra or a small detached fragment of bone. In other words, the röntgen findings may be of no greater significance, and merely serve to show the location of the injury. This is not an uncommon experience in cervical spine injuries, where the röntgenographic evidence of traumatism to the spine frequently bears little relation to the severity of cord injury. On the other hand, the röntgenogram may show a serious fracture with marked permanent displacement, and there may be little or no cord disturbance. Such injuries are usually found only in the upper cervical region. Extensive injuries in the lower cervical spine are invariably serious.

Uncomplicated dislocation in the first or second cervical vertebra, especially when unilateral, may be best demonstrated in an antero-posterior röntgenogram made through the wide-opened mouth. With this exception, most injuries of the cervical spine are most satisfactorily shown by lateral views.

Pure dislocations are quite common at the lumbo-sacral articulations and may be unilateral or bilateral. They may easily escape notice unless stereo-röntgenograms are made. In unilateral luxations the spinous process of the fifth lumbar vertebra may be found out of line in the flat plate, and a considerable difference may be observed in the appearance of the articulations on the two sides. In bilateral luxations the body of the fifth lumbar vertebra may appear to project below the line of the promontory of the sacrum in the antero-posterior view. A lateral view of this region is difficult to obtain and invariably lacks clear detail, but if it shows enough to reveal the body of the fifth vertebra projecting abnormally beyond the anterior aspect of the body of the sacrum it serves its diagnostic purpose.

Aside from these remarks injuries of the spine may be discussed from the standpoint of fracture, which from the röntgenologic point of view can be most conveniently grouped upon an anatomical basis—namely, those of the spinous processes, transverse processes, posterior arches, and bodies. The same rules apply to röntgen examinations of the spine as to fractures elsewhere—two views should be made in directions perpendicular to each other whenever possible, and stereo-röntgenograms should be made whenever they are likely to give additional diagnostic aid. The röntgenologist should bear in mind that movement of the patient may be fraught with serious consequences in recent injuries, and the surgeon should consider the difficulties that naturally beset his confrère and render what assistance is possible in advising him of the seriousness of the injury and eliminating obstacles to a thorough examination. The patient should be examined before being placed on a water or air mattress. In injuries of the lower thoracic and lumbar region the gastro-intestinal tract should be as empty as possible. The röntgenologist may materially assist the surgeon in a subsequent operation by correctly identifying some landmark in the injured region. This may be done by making an indelible mark, as with nitrate of silver, over a spinous process and then fastening a piece of lead wire over this mark with adhesive plaster. The surgeon can then readily determine from the röntgenogram which vertebra has been marked and its relations to the seat of injury.

Fractures of the spinous processes must be determined in almost all cases from lateral röntgenograms. Fortunately they are more common in the cervical region where lateral views are most readily obtained. In the thoracic vertebræ the diagnosis is very uncertain because of the difficulty of making lateral or oblique views that will satisfactorily show these processes. In the lumbar region the diagnosis again becomes comparatively easy provided there are no serious difficulties to be encountered in obtaining lateral röntgenograms.

Fractures of the transverse processes are far more common in the lumbar vertebræ than elsewhere and can usually be demonstrated readily in the easily obtained antero-posterior view. A normal appearance simulating fracture is quite common in one or both transverse processes of the first lumbar vertebra, and is due to a separate center of ossification in an attempt to form a lumbar rib.

Fractures of the posterior arches are particularly important because of their close relations with the cord. The accurate determination of their presence and the extent of encroachment upon the cord requires a lateral view, and even this frequently fails in its purpose even in a clear röntgenogram. The amount of cord pressure exerted by the laminæ may be suggested by the extent of forward displacement of the body of a dislocated vertebra, which is easily demon-

strated in the cervical spine and usually in the lumbar region also, if the patient is not too stout and can be placed in an advantageous position. In the thoracic region satisfactory lateral or oblique views except in the lower segment are far more difficult, especially in cases of recent injury. If the laminæ of the lumbar vertebræ are broken in the region of the articular processes, the lesion may be detected in a clear antero-posterior plate, provided there is ample intervertebral space and no decided deformity from luxation.

Fractures of the vertebral bodies may be divided into two groups—those in which a well defined fracture exists and those in which there has been merely a crushing of the cancellous structure, the appearance of which may vary in extent from a very slight alteration in shape to a decided perpendicular narrowing of the body, especially the anterior portion. The latter type of injury is usually found in the lower thoracic and lumbar regions. The former or true fracture can usually be detected in an antero-posterior view, except frequently in the cervical region, where the lateral view is almost always essential. The antero-posterior view does not, as a rule, however, indicate the full extent of the injury, and it certainly does not convey a correct impression of the amount of cord encroachment in case there is an accompanying dislocation or fracture of another portion of the vertebra. The uncomplicated crushing fracture of the body may escape detection entirely in the antero-posterior view, but may be easily demonstrated when examined laterally, even though the röntgenogram is lacking in detail of all other portions of the vertebræ. Certainly, a negative diagnosis in a spinal injury cannot be accepted as final unless both antero-posterior and lateral plates fail to demonstrate any lesion.

The treatment.—The general symptoms of injuries to the cord by bullets or by cutting instruments are similar to those which are due to spinal fractures, with the difference that the wound is always a compound one and, therefore, the danger of infection is added. With the high velocity of the modern rifle bullet, however, the frequency of infection has become much less, and many wounds heal without suppuration. The injury from the high-velocity bullet may be very exactly limited to one part of the cord and portions of the fiber tracts, or entire tracts may be divided with almost anatomical exactness, just as if experimentally produced. On account of the great laceration of the soft tissues, wounds that are caused by explosive projectiles are very often infected.

The indications for treatment will depend entirely upon the diagnosis of the completeness or incompleteness of the injury to the cord. Whenever, after an injury there is complete paralysis below the level of the lesion with complete sensory loss (including the vibratory sense), absence of all tendon and cutaneous reflexes, and loss of con-

trol of the bladder and rectum, the lesion must be considered a complete one until symptoms which show that the lesion was not complete make their appearance. Excepting in the case of injuries below the level of the first lumbar vertebra, these patients should never be operated upon, and conservative treatment is indicated until some of the reflexes return or there is other evidence that the injury to the cord has not been followed by a complete transverse lesion. Injuries to the roots of the cauda equina should usually be operated upon, as it may be possible, by suture of the nerve roots, to obtain a regeneration of the motor roots of the cauda equina. In some instances severe root pains from compression of posterior nerve roots by dislocated or fractured bone, may make an operation imperative, but in most instances of complete transverse division of the cord, there is no pain at all. The presence of fragments of shrapnel or high explosive in the wound or extensive laceration of the dura may call for operative intervention.

The spinal cord symptoms are due either to injury or compression of the cord by fractured bone, to actual division of the cord by the bullet or high explosive, to hemorrhage into and around the cord substance, to acute edema, or to secondary inflammation of the meninges.

The presence of the bullet in the spinal canal as demonstrated by the X-ray does not always mean an irremediable cord lesion. If the symptoms begin to improve within a few days or weeks, a laminectomy should be performed, and the bullet removed without delay. If the bullet passed through the canal, the X-ray may demonstrate this fact and may give the surgeon an idea as to whether the bullet has passed through part or the whole of the thickness of the cord. The symptoms of a transverse lesion at a definite level may be due to concussion. Many of these patients begin to improve after a few days. Similarly, a spinal concussion may be seen when the projectile has struck a spinous or transverse process or if a bullet has penetrated a vertical body. There are many combinations of spinal concussion and direct spinal injury; it is very difficult, therefore, to give exact indications and contra-indications for operative interference in bullet wounds of the spine. The following general rules may be laid down:

1. When the symptoms are those of a complete transverse lesion of the cord, it is inadvisable to perform an operation, unless the nature of the wound or the presence of a foreign body demands operative intervention. In the latter case, the treatment is that of a lacerated wound in any part of the body, excepting that if the dura has been injured, especial care must be taken to prevent an infection of the meninges. It is certainly very rare for effused blood or the mere pressure of a foreign body (splinter of bone, bullet, shrapnel ball, etc.) to cause

the symptoms of a *complete* transverse lesion. Some sensation or power, or a few reflexes will persist in most of the patients.

2. In incomplete lesions of the cord operative interference is often required at once or within a few days or weeks of the injury, unless the symptoms are so slight that improvement may be expected without surgical intervention, unless there are complicating injuries of the thorax or abdomen, which contra-indicate surgical interference or unless the patient's condition is so poor that any operative interference is useless.

The non-operative treatment.—An individual with a spinal cord injury should always be kept as quiet as possible, and should not be transported for the first few days, if this can be avoided. The shock is often surprisingly slight and is most apt to be encountered in patients with cervical injuries. The surgeon should always search carefully for injuries to the abdomen or thorax, as these are very frequent with bullet wounds of the spine. Morphin as a sedative is often necessary, but it should be given in very small doses in cervical cord injuries, as these patients are often very susceptible to the drug.

If the injury affects the cervical cord, the upper portion of the vertebral column should be immobilized by bandaging the patient to a *well padded* posterior splint. For this purpose a board about 12 inches wide and 4 feet in length should be used, and the splint should be applied so as to project 6 or 8 inches beyond the patient's head. By this means the patient can be raised without disturbing the region injured. In other parts of the cord, immobilization is necessary only when there has been extensive bone destruction.

The following notes on *fixation methods in fractures of the spine* have been prepared by the orthopedic section and are quoted in full:

"Adequate fixation of the spine continued for a sufficient length of time greatly lessens the danger of the incidence of late pressure symptoms or of bony deformities.

"Fixation should be secured by recumbency in all cases of established vertebral fracture during the period of acute reaction following the trauma. The duration of bed treatment will vary from a week or two in the milder cases to many months in those cases with marked dislocation and deformity accompanied by compression of the cord. The necessities of war occasionally interfere with recumbent treatment for mild cases. These should be immobilized so far as possible by means of braces or casts for transport. Ambulatory apparatus is also needed in the convalescent phase of the more serious cases to provide for the support of muscles suffering from the atrophy of long disuse.

"*Position.*—Support of the normal curves of the spine is always essential. Over-correction may be indicated for the relief of pressure

and the prevention of deformity. Lateral deviation must be borne in mind and corrected or avoided by proper appliance.

"*Location of lesion.*—The required fixation varies with the location of the lesion. High cervical fractures may often be treated with fixation of the head alone. Fracture of the lower cervical vertebræ and the upper half of the dorsal spine usually require apparatus for immobilizing the head as well as spinal fixation. From the mid-dorsal region to the fourth lumbar vertebra fractures may often be held by a jacket alone which should include and stabilize the pelvic girdle. Lower lumbar, sacral, sacro-iliac, and pelvic fractures should be immobilized by an appliance to include the back and one or both thighs.

"Many cases of simple fracture of the spine occur as a result of military operations from accidental causes, as the caving in of trenches and dugouts, being hit by flying sandbags or trench timbers, and from being thrown by the concussion of exploding shells. But the greater number of cases encountered as battle casualties are compound fractures, and very often show, in addition to considerable wounds, very extensive comminution of bone. Many wounds can not be closed primarily, and prolonged septic dressings are required. This complicates the problem of fixation to a marked degree. Provision must be made in whatever appliance is used to avoid pressure on the wound and to render the dressing accessible. On account of the difficulty in meeting these provisions, plaster of Paris is little used for these cases except as a means of transport.

"While it is impossible to discuss at length a complete series of the methods available for immobilization of the spine, the following appliances are mentioned on account of their simplicity, efficiency, and availability. Emphasis is laid on only special points of technic which have been found of standard value; the details of management of any individual case must necessarily depend on environment and materials locally procurable.

"*Head traction.*—There are three simple methods of value for providing head traction. The first consists of a band of 1-inch webbing with buckle which fastens about the head, passing just above the eyebrows in front and below the occipital protuberance behind where it is well to pad it with a fold of cotton flannel, as it is at this point that it takes its chief traction bearing. Side straps are sewed to this band about the midtemporal region and are carried upward to a spreader over the head, which is in turn attached to a cord and weight carried over a pulley at the head of the bed.

"The second method is by means of a strip of unbleached cotton 5 inches wide and 40 inches long. This is torn from both ends toward the center so that one strip shall be 2 and the other 3 inches wide and leaving an untorn central portion about 6 inches long. The

wide border of this central portion is carried under the chin and the ends brought upward in front of the ears. The narrower border is carried in front of the chin and its corresponding ends backward under the ears, crossed below the occipital protuberance and then brought upward and forward to the other ends to which they are tied just above the ears. Traction is taken from the point where the strips are tied at the sides of the head.

"A third method is to use a webbing band as in the first method over the brow, but to substitute for its posterior half a sandbag shaped much like a horseshoe, the bend of the shoe passing below the occipital protuberance and traction being taken from the two legs of the horseshoe coming upward back of the ears. This has the advantage of providing secure traction and at the same time immobilizing the head by preventing rotation.

"*Head and leg traction.*—By adding to the above head traction the ordinary Buck's extension traction straps to the legs, fixation is rendered much more complete. Adhesive strips applied to the legs are useful, but the more commonly used flannel bandage glued to the skin by means of specially prepared glue will be found more comfortable, rather less apt to irritate the skin, and more durable.

"*The Thomas collar.*—The Thomas collar is a most useful appliance for injuries of the cervical spine. Used alone it partially immobilizes during recumbency, and combined with head and leg traction acts as a very satisfactory method of complete fixation. In cases of mild injury or during convalescence, it is a useful ambulatory appliance. It may be improvised easily from materials always at hand.

"*First method.*—The Schanz collar is made as follows: A 5-inch bandage of unabsorbent cotton is wound about the patient's neck while the head is held in the desired position. After a few turns a gauze bandage is applied to compress the cotton. Further layers of cotton and gauze are added, the gauze being drawn tighter as the thickness of the collar increases. It should be made very bulky, filling the entire submental space, and as it loosens with wear more cotton may be tucked under the edges of the collar, and a tighter bandage applied to maintain its adjustment.

"*Second method.*—A strip of flexible cardboard such as is used for laundry boxes, 18 by 6 inches in size, serves for a framework. This is hollowed out along the middle of its upper border to accommodate the chin and similar hollows are cut from its lower border where it passes over the shoulders. The upper edge is cut down behind to fit below the occipital protuberance. Two pieces of bandage or two buckle straps are laid lengthwise on this board, their ends protruding beyond the ends of the board. These are used to fasten the collar on after completion. Sheet wadding or picked oakum is

wound thickly about the frame and bandaged on. The collar should be shaped to the patient's neck during the winding and may be raised at any part to secure over-correction, if desired. The tightness of fit is determined by drawing up on the bandages or buckle straps provided for securing the collar in place.

"*Third method.*—A more durable collar may be made by using plaster of Paris over thick cotton wadding. By protecting the edges with felt, a comfortable and more permanent support is provided. Fixation is not so complete, however, as there is no means of adjustment, and after the first day or two a certain amount of play will develop from compression of the padding material.

"*Plaster of Paris.*—Plaster of Paris is used in two ways as a means of fixation, as casts and in the form of plaster beds. Great caution must be used in handling the patient, and a proper table is quite indispensable. In addition to sheet wadding and stockinette, adaquate felt padding must be applied over all bony prominences, the chin, the occipital protuberance, spinous processes, scapulæ, acromions, crests of the ilia and anterior superior spines. This is of special importance on account of the readiness with which decubitus develops in paralyzed cases. When the head is included, iron straps, one-eighth inch thick and a half inch wide, bent to fit the neck and cranial curves, will aid in its application and strengthen the cast. These are also useful in applying jackets and especially to reinforce the groin in spicas.

"Plaster beds are difficult to apply, especially during the acute conditions following trauma. As a means of transport they furnish an excellent fixation appliance. They are most easily applied as casts, and cut along a lateral plane when set, the upper half being discarded. Or the patient may be turned on his face, the chin resting symmetrically on a pad, and the plaster laid on over proper felt and cotton protection. In this case the cast is carried well down over the sides of the body, bandaged on while still wet, the edges being trimmed later, and preferably bound with adhesive plaster. Once a day the patient in a plaster bed should be turned, the bed lifted off, and the skin carefully rubbed and powdered.

"*Jackets of other material.*—Other materials are used instead of plaster for making jackets. Spinal corsets of celluloid and leather are more expensive and require skilled labor in their production, but they are cleaner, more durable where there is need for long-continued support, and are often more comfortable. They require a plaster cast for a model, and are fitted over a mold poured in the cast. Regular corsets of cloth reinforced by the usual steel or heavier metal uprights are of service in certain cases, especially as convalescent supports.

"*Metal braces.*—The usual Taylor back brace modified to suit the individual case is often of service. In high lesions a jury-mast or collar may be added.

"*Bradford frame.*—The Bradford frame is the most important single appliance for the care of cases of fractured spine. It has been standardized for regular army supply so that it will fit any regulation cot, and also the standard stretcher. The present narrower width gives opportunity for fixing the patient by means of the swathe and cross straps familiar in the treatment of children with spinal caries. It provides a ready method of transport without injury or discomfort to the patient, and is a most useful aid in the nursing care of these difficult cases. Correction of deformity may be treated by bending the side bars of the frame in such a manner as to secure moderate hyperextension of the spine. Such padding as is necessary to preserve the normal spinal outlines is easily held in place by attaching it to the cover of the frame. An air mattress will always be available and is of very great service in these cases in the prevention of decubitus. Rubber rings are most useful for the same purpose.

"*Preparation of the bed.*—In cases where paralysis is complete the difficulties in the way of putting on a cast or supplying a plaster bed are so great that all splinting is omitted, and the patient merely placed in proper position in a suitably arranged bed. Most springs sag so badly that fracture boards should be provided. On them an air mattress gives the safest support. This should not be fully inflated. A good rule is to blow it up enough to allow the hand, with the body weight resting on it, just to press through the mattress. When the weight of the injured man is distributed over a mattress so inflated it will not be compressed too much at any one spot, but will adapt itself to the pressure points of the body. It is well to add a firm pillow about 2 inches thick under the lumbar region, and a pillow also under the knees to maintain a few degrees of flexion and prevent the tendency to hyper-extension resulting from relaxation of the paralyzed muscles. When necessary, over-correction may be provided by supplying more pillow support at the points indicated. Sand bags placed along the body and limbs will serve to steady the patient by preventing rotation. The heels should be supported in ring-shaped pillows to lessen the danger of pressure necrosis.

"It must be remembered that deformity develops with great rapidity in a paralyzed bed patient, and to prevent foot-drop, provision should be made to support the foot at right angles. This may be done by means of a foot splint, but to avoid danger of undue pressure the method employed in the Balkan frame apparatus is serviceable. A piece of 3-inch flannel bandage is glued to the sole of the foot, and to that a cord and weight is attached and slung over a

pulley placed on the overhead bar, slightly mesial to, and a foot or more up the leg from the foot. By means of a weight of two or three pounds this secures a permanent position of slight inversion and moderate dorsal flexion. Cradles should be adjusted to prevent pressure from bed clothes on the skin of the knees and toes. This may also be accomplished by pinning the blankets to bandages hung from the overhead bar of the Balkan frame in such a way that the bedding is lifted just clear of the patient.

"One of the old type Bradford frames, which is wider than the patient, may be used in conjunction with the air mattress and greatly simplifies nursing care. By lacing the cover loosely, it does not interfere with the efficiency of the mattress, but does add a means of lifting the patient for dressing and other purposes as well as simplifying his transportation to operating theater or X-ray room, when necessary."

Whenever possible, as has already been stated, the patient should be on an air or water bed, and for the first few days should lie flat on his back. Good nursing is important, so that decubitus shall be prevented. All parts of the body, especially the sacrum and iliac crests, should be protected by soft pillows or rings. It is usually necessary to place a cotton pad between the knees of the patient as they lie against each other, and also to protect the heels by small rings of cotton. Hot-water bags should never be used on any part of the body below the level of the lesion.

As has been mentioned above, bedsores can often be prevented by careful nursing. The smallest red area from pressure of the bedclothes should be carefully watched for. Especial care must be taken by the nurse that the bedclothes are not creased and that there are no crumbs of bread or other particles of food under the patient. After forty-eight hours, if there be no contraindications, the patient's position should be frequently shifted. If bedsores do occur, they must be treated according to general surgical principles.

The care of the bladder and rectum.—The care of the bladder and rectum is of the utmost importance. Most of the patients are constipated unless they have received a laxative, and the bowels should, therefore, be moved by daily enema or by small doses of a mild laxative. If there is retention of the urine, catheterization should be done with the greatest attention to asepsis. Even at the best, cystitis will occur sooner or later. With the first evidence of cystitis a permanent catheter should be inserted for a few days, and with this procedure the possibility of an acute epididymitis should not be overlooked. The bladder should be washed out daily with a weak permanganate of potash solution or boric-acid solution or a 1-10,000 nitrate of silver solution. It is not advisable to allow a patient's bladder to become over-distended until it overflows, as very aggravated cystitis

often result from this cause. In some instances, in patients with complete transverse lesions and incontinence, a water-tight fistula, according to the Witzel principle, may be made by suprapubic cystotomy, but this is rarely necessary. Those patients who have incontinence usually need only to have the proper receptacle so that the bedclothes shall not become soiled. Every patient with a spinal injury, who has to be catheterized or who has an alkaline cystitis, should be given large doses of urotropin; from 30 to 60 grains a day are recommended after the urine has been acidified, but the urotropin must be stopped as soon as there are signs of renal or bladder sphincter irritation as evidenced by the presence of blood in the urine. In spite of the best of care, however, very many of those with a severe spinal injury and inability to empty the bladder, develop a cystitis, and in them the danger of an ascending infection of the kidneys is very great.

In patients with an incomplete spinal lesion, cystitis is not as apt to occur, and in them care in catheterization will be followed by satisfactory results. Every patient in whom there is an incontinence of urine should be catheterized at least once in order to make certain that the apparent incontinence is not really an overflow. Complete incontinence occurs most often in transverse injuries of the cord or in partial injuries of the lumbo-sacral segments.

Individuals who have an injury to the roots of the cauda equina most often have retention of urine, although many of the patients are able to partly empty the bladder by contraction of the abdominal muscles. Catheterization once in every 24 to 48 hours is usually necessary, and whenever catheterization is done, the bladder should be washed out with weak permanganate of potash or boric-acid solution or 1-10,000 of nitrate of silver solution.

After lesions of the cord in the mid and lower dorsal region abdominal distention is very frequent. Not so rarely a very marked and, to the patient, very distressing abdominal distention occurs, which can be relieved only partly by cathartics or enemata and which usually persists for several days. The patient is unable to pass any gas, the abdomen becomes enormously distended, and vomiting occurs, so that the patient seems to present the clinical picture of an acute intestinal obstruction. The symptoms, which are probably due to a disturbance in the superior splanchnic nerve, usually subside within a few days. The distention can sometimes be controlled by a dose of pituitrin given hypodermatically, but in most instances the patient's discomfort has to be relieved by morphin until the distention subsides.

Wounds of the back, whether or not they are complicated by fractures of part of the vertebræ, must be treated on general surgical principles. If the patient is seen early, every effort must be made to

prevent infection. If seen later, infection must be combated by every means at our command. In many lacerated wounds of the back, the Carrel-Dakin treatment is very useful in controlling infection in the paravertebral muscles.

If there is leakage of cerebrospinal fluid, the care of the wound must be redoubled, and the skin around the wound kept aseptic by painting it with tincture of iodin at each dressing.

Massage and electricity to paralyzed muscles or limbs can usually not be begun until several weeks have elapsed. In patients with receding symptoms of cord injury, this treatment is very important, because it may prevent those atrophies, stiff joints, and contractures which often delay recovery for many months. Attention to the paralyzed and overstretched muscles is very important, and if a number of vertebræ have been fractured, the spine must be supported by proper apparatus or splints (see "Fixation Methods in Fractures of the Spine," p. 265).

The operative treatment.—A careful neurological and X-ray examination must always precede and make the indications for operative interference, and three questions of diagnosis must always have been answered. The lesion must have been located, and the degree and kind of lesion approximately determined. While X-ray examinations should never be omitted, the results are often unsatisfactory; the neurological examination should be most relied upon for establishing the location of the injury.

The structure of the vertebral column should be carefully studied by those who desire to do successful spinal surgery.

The spaces between the cervical and between the upper dorsal spinous processes are of considerable size, and there is no trouble in removing each spine at its base with appropriate rongeur forceps. The middle dorsal spines, however, overlap each other so much that it is not so easy to isolate each spinous process for its removal. In the lumbar region, again, the spines, though very thick and short, are easily removed.

The removal of the spinous processes and laminæ in the cervical and greater part of the dorsal regions will lay open the spinal canal widely. In the lowest dorsal and lumbar regions the canal is more deeply placed, and only the removal of the laminæ well out to the articular processes will give a free exposure of the spinal canal and its contents.

The muscles that surround the cervical and dorsal vertebræ are easily separated from the spines and laminæ, but in the lowest dorsal and the lumbar vertebræ the prominences of the transverse processes interfere somewhat with the retraction of the muscles. In the lumbar region, also, the muscles are thicker and more deeply placed between bony prominences.

The ligamenta flava are much thicker in the lower dorsal and lumbar regions and must often be separately excised.

The outer surface of the dura is covered by a layer of fat, which must frequently be stripped off from the membrane and removed before the dura is exposed to view.

Ordinarily it is necessary to remove the arches of at least three vertebræ in order to obtain sufficient exposure, and it is better to take away one arch too much than one too little.

It is important to remember that the segments of the cord are always on a higher level than the correspondingly named vertebræ, and that the surgeon must go at least two vertebræ higher than the correspondingly named segments. Thus, to expose the sixth dorsal segment it is necessary to remove the arches of the third and fourth dorsal vertebræ. When the X-ray has demonstrated a foreign body which is to be removed, the operative area is, of course, mapped out for the surgeon. But in bullet wounds of the cord, the cord injury is sometimes at a much higher or lower level than the point of entrance of the bullet, and the surgeon must be guided by the level symptoms rather than by the wound.

The spinous processes desired should first be located both by counting downward from the seventh cervical (vertebra prominens) and by counting upward from the fourth lumbar (on a level with a line connecting the highest parts of the iliac crests). The incision is made over the spines and rapidly deepened on one side of the spinous processes. The scalpel must be kept as close as possible to the spines, so as to divide the muscular attachments near the bone. If the bullet wound is in the line of the incision, it should be carefully circumcised and the skin around it freely excised. The separation of the muscles is completed with a broad elevator and the oozing controlled by a gauze packing. The same procedure is then accomplished on the other side. The bleeding is seldom profuse, rarely requiring the use of artery forceps, and is controlled by the packings. There are a few large blood vessels which must occasionally be caught with forceps. If the vertebral arches have been fractured, the fragments of bone must be removed with great care. Especial care must be taken if splinters of bone have perforated the dura. In the latter case, the splinters should not be removed until the dural sac has been freely incised and the relations of the bone splinters to the cord determined. The packings are removed after a minute or two and the wound edges spread apart with an automatic retractor. The retractor not only controls all oozing from the muscles, but it gives an excellent exposure of the field of operation.

Considerable blood and clots are often encountered as soon as the vertebral arches have been removed. This blood should be care-

fully sponged away until the dura is well exposed. As soon as the bone has been removed the cavity is packed with gauze wrung out in hot solution and by this means all bleeding is controlled. A little oozing from around the divided laminæ may persist. This can usually be controlled by traction on the flaps of the dura after this membrane has been incised.

Exploration of the spinal cord.—Those inexperienced in spinal surgery sometimes find it difficult to recognize the nature of the abnormal conditions which are found. A few words upon the normal and pathological appearances of the cord and membranes are therefore permissible.

Normally, the outer surface of the dura is of a bluish-white color and is smooth and glistening. A reddish or brownish discoloration is due to adjacent bone disease or to an inflammatory process in the dura itself. To the examining finger the membrane feels soft, and it is easy to recognize that it is thin and that there is fluid underneath it. If the finger is gently passed over its surface, one can feel if the membrane is thickened.

After the dura has been incised, the arachnoid will be seen as a clear transparent membrane, which is not adherent to the smooth shining inner surface of the dura. Adhesions between the inner surface of the dura and the arachnoid are pathological, and any cloudiness or thickening of the arachnoid is an evidence of the disease. Frequently, small calcareous plaques occur in the arachnoid; these have no pathological significance and can be peeled off without difficulty. The surface of the cord is of a creamy-white color and is covered by a fine network of blood vessels. If the vessels are very large and tortuous, it means that there is either an inflammatory process within the cord substance or in the pia or that there is a pressure upon the vessels of the cord at a higher level than that exposed. In myelitis and meningomyelitis the cord is often of a reddish color and is covered by numerous dilated blood vessels. When there is an obstruction to the return flow of the blood in the posterior spinal veins, the appearance of the cord below the level of the compression is altered. The normal creamy white color is preserved, but the veins are dilated and tortuous, so that the swollen vessels stand out very prominently. After some experience, the surgeons should be able to differentiate between a hyperemia of the cord due to inflammatory disease, and a venous congestion from compression of the spinal veins.

In order to explore the anterior surface of the cord, it is advisable to grasp a slip of the dentate ligament with mosquito forceps, to cut the slip at the dura, and then to raise the cord by traction on the ligament. Traction on a slip of the dentate ligament is the

best method for rotating the cord in order to expose its anterior surface.

Pathological conditions.—*Fracture of the spine without injury of the dura.*—In these patients there is always considerable blood on the outer surface of the dural sac. The membrane may be depressed by dislocated fragments of bone. All loose fragments of bone should be removed with care. If they are still attached to the vertebral arches, they should be bitten off with rongeur forceps. Foreign bodies should be carefully searched for and removed. In the removal of jagged fragments of high-explosive shells, it is better to enlarge the wound than to attempt to free the fragment by rough manipulation. If the vertebral muscles are much lacerated, loose shreds of muscle tissue should be excised, the entire wound sponged out with weak iodin solution (tincture of iodin diluted with an equal amount of alcohol) and the wound then partly closed with free drainage. If the cord symptoms are well marked, the dura should be incised and the fluid blood or clots removed by gentle irrigation with a saline solution. The cord should then be examined. The injury to the spinal cord is often a grave one. In these fresh fractures there is often a large amount of blood clot in front of the dural sac. The clots can be washed away by a stream of saline solution or by the careful use of small sponges. Fractures of the body of a vertebra are very frequent, and are complicated by considerable extravasations of blood from the venous plexuses, which exist on the posterior surfaces of the vertebral bodies.

Fracture of the spine with laceration of the dura and injury to the cord—The appearance of the crushed cord—Hematomyelia.—If the dura has been injured, a marked cord lesion is to be expected. If there has been marked comminution of the arches of the vertebra, fragments of bone may be found within the dural sac. They must be removed with great care. As soon as the blood or clots have been evacuated, the contused or crushed cord becomes visible. It may appear of normal size and color but abnormally soft and somewhat depressed at one spot. At other times, a distinct transverse depression with a yellowish or reddish-brown discoloration is observed. Most frequently the cord is swollen and hemorrhagic. Above and below the most affected area, the cord appears soft and perhaps flatter than normal. The most extreme care must be taken not to touch these softened areas, because the smallest amount of manipulation is harmful. After all clots and fluid blood have been washed away, the dura must be tightly closed by a continuous suture of fine silk. If there is a defect in the dura, a piece of fascia may be taken from the fascia lata of the thigh and transplanted, or the defect in the dura may be covered with a piece of Cargile membrane. If fragments of

clothing or other foreign body were removed from inside of the dura, the exposed cord and the inner surface of the dura may be gently sponged off with a small cotton swab and 1-1,000 bichlorid of mercury solution.

If the injury is a very recent one (within the first 24 hours) and the cord is found much swollen and discolored over a localized area, it is advisable to make a small incision in it at or near the posterior median septum. This incision must be made with a fine cataract knife and should be no more than 1 to 2 mm. in depth and 1 to 2 cm. in length. The object of this incision is to allow of drainage of edematous fluid and blood from the cord substance into the dural sac. The edema which occurs in a contused cord is of a very destructive nature and may contribute to an acute transverse softening at the affected level. This incision of the cord must be done with so much care and precision, that it should be performed only upon the strictest indications and by a surgeon who has had experience in intramedullary spinal surgery. Needless to add, the cord must never be grasped with an instrument or with the fingers.

In these swollen and contused cords, the removal of the laminæ must be wide enough to obtain the full decompressive effects of the operation, and it should be a general rule rather to remove too much than too little of the laminæ.

If the cord is not much swollen or discolored and if the surgeon suspects an intramedullary collection of blood, this blood should be aspirated. The spinal cord can be punctured with a needle with entire safety, provided that only the finest aspirating needle is used and care is taken not to injure even the smallest vessels. In doing the aspiration, it is advisable to make the puncture near, but not through, the posterior median septum. The cord must be carefully steadied, as the pia offers some resistance to the entering needle. A slip of the dentate ligament is grasped on each side with fine mosquito forceps and held in place. Extreme care must be taken that the direction of the needle is held constant.

If clear fluid is obtained by aspiration, a small incision, a few millimeters in length, is made in one posterior column into the cavity. The edges of the incision usually separate somewhat, so that the opening in the cord will remain patent, and the fluid will drain into the subarachnoid space.

It is hardly necessary to emphasize the fact that an incision in the cord in any condition should be made only after the most careful consideration of its advisability, and should be performed only by a surgeon who is experienced in this line of work. The utmost care is necessary in order to cause no serious lesion, and the very delicate procedure must be accomplished without the minutest bleeding.

The presence of intradural or intramedullary bullets or splinters of bone.—To remove a bullet or fragment of bone that lies in close contact with or has partly penetrated the cord requires the most careful and delicate manipulations. Otherwise a partial lesion may be transformed into a complete cord lesion.

After the surgeon has definitely established the relations of the foreign body to the cord, he must decide how this foreign body is to be removed so as to cause the minimum additional trauma to the cord. The foreign body should be carefully grasped with forceps and slowly manipulated until it has been delivered. No force should ever be used. If the bullet or bone splinter lies in front of or against the side of the cord, a slip of the dentate ligament should be grasped with mosquito forceps and cut at its attachment to the inner surface of the dura. The cord can then be raised or rotated to one side by traction on the ligament.

If a bullet has become mushroomed or the bone splinter has irregular jagged projections, these had best first be bitten off with small sharp rongeurs before the foreign body is removed.

Especial care must also be taken when a bullet, shrapnel ball, or fragment of high explosive is found to be embedded in the body of a vertebra and to compress the cord from the side or from in front. Considerable force is often required to loosen such a body, and the surgeon must be most careful that no pressure beyond what is absolutely necessary is made upon the cord during the manipulations. The lumbo-sacral cord is especially vulnerable and is sensitive to the smallest amount of handling. It is a good general rule to consider that every time a part of the spinal cord is touched harm is done. The surgeon who has this point in view will reduce the handling of the cord to the minimum.

Incised wounds of the cord.—It is useless and harmful to attempt to suture a partly divided spinal cord. Experience and experimental investigations have shown that, excepting for seeming microscopic efforts at regeneration, no true regeneration in the cord is possible. Nor should the ends of divided nerve roots be sutured together. There is no functional regeneration of the anterior or posterior nerve roots inside of the dural sac, excepting in the roots of the cauda equina.

Injuries to the roots of the cauda equina.—While it is a general rule in the surgical treatment of spinal cord injuries to control even the smallest amount of bleeding before the dura is sutured, this is especially important when the roots of the cauda equina are concerned. If blood clots remain behind, adhesions form and the patients are very apt to suffer root pains which may require a second operation for their relief.

After all bleeding has been controlled and foreign bodies removed, the roots of the cauda are carefully examined. At their origin from the conus the lumbar and sacral roots lie close together, and if the roots could be identified in this situation, only two or three vertebral arches (twelfth dorsal, first and second lumbar) would have to be removed. Attempts have been made to identify the nerve roots at the conus instead of at their foramina of exit from the dural sac. These efforts have not given very satisfactory results, because in most instances the nerve roots can not be distinguished from each other with certainty. Occasionally, however, if all of the posterior roots are raised up on a probe, the nerves can be distinguished from each other. The fourth and fifth lumbar and the first sacral roots are much larger than the second sacral or the upper lumbar roots. The posterior is usually larger than the anterior root and lies behind and to the outer side of it. In order to free the posterior root, the entire nerve is first raised up on a strabismus hook. The line of separation between the motor and the sensory roots is usually found without difficulty.

For the identification of the roots, it is a good plan to remember that the first lumbar root runs over the lower end or "fork" of the dentate ligament. After the ends of divided nerve roots have been identified, they should be carefully sutured together by perineural sutures of fine silk. The dural sac should then be closed in the usual manner.

The treatment of infected wounds of the back and spine.—If a wound in which the dura has been lacerated becomes infected, the symptoms of a rapidly advancing meningitis usually set in early. The infection, usually due to the ordinary organisms of suppuration, generally spreads with great rapidity; and, no matter how great the effort to control the infection, almost always ends fatally. Occasionally, however, even such a patient may be saved by the wide drainage of the wound and large doses of urotropin (60 to 120 grains a day).

Free drainage of the wound is also necessary in those patients in whom the dura is intact, but in whom a general wound infection has occurred. If the infection is not controlled early, by drainage with tubes, by Carrel-Dakin treatment, etc., the infection may spread into the meninges. Some of these patients develop an extradural abscess, with high fever, profound prostration, and (if they have not a complete transverse lesion) a marked hyperalgesia in the lower extremities. The hypersensitiveness is often so extreme that the smallest movements or the slightest handling of the limbs, or even the weight of the bedclothes causes excruciating pain.

General considerations relative to the aftercourse of patients upon whom a laminectomy has been performed.—If there are no complica-

tions from the anesthesia and if the wound healing is aseptic, the convalescence of a patient who has undergone a spinal operation, should be a very uncomplicated one. For several days the patients complain of much pain in the back, which is made worse by movements of the limbs or head, and anodynes and hypnotics are usually required.

Constipation is frequent, and the bowels are best emptied daily by enemata.

The lower limbs must be supported on pillows. If sensation and power have not been completely lost in the lower extremities, the least movement of the limbs will cause the patient much pain in the back. This is due to contraction of the paravertebral muscles when the patient attempts to move the lower limbs. The pain usually disappears within a few days.

Some of the patients have a rapid pulse (120 to 140) for a few days after the operation. This is especially apt to be the case when the laminectomy has been performed in the cervical region.

A rise of temperature to 102° or 103° F. is very frequent after a spinal operation in the cervical and upper dorsal regions, and occurs most often in the patients in whom the dura has been opened. The temperature usually falls to normal within two or three days. After cervical laminectomy, the temperature may remain several degrees above the normal for a week or more.

The knee-jerks are often lost for 12 to 24 hours after the operation, and the ankle-jerks are often much depressed for several days. These changes are probably connected with changes in the intraspinal pressure due to the loss of cerebrospinal fluid and to the entrance of air into the subarachnoid space. They do not occur in those operations in which the dural sac is left unopened.

The general principles of the treatment after the operation of laminectomy have been considered in the section devoted to the non-operative treatment of spinal injuries. Unless there are special indications, a laminectomy wound should not be dressed for 7 to 10 days, when the sutures are removed.

THE OPERATION OF LAMINECTOMY.[1]

Anesthesia.—In most instances the operation is carried out under general anesthesia, although occasions have arisen when I have thought a general anesthetic might be undesirable, and under such circumstances, referred to below, I have been able to complete the operation under local anesthesia. With these exceptions ether is used in my clinic and administered by the endotracheal insufflation method

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of Meltzer. The apparatus designed by my associate, Dr. George P. Muller, has proven in every respect satisfactory; it is less complicated and less expensive than many others on the market. The tube is introduced with the Jackson laryngoscope, and after the patient has been placed on the operating table no concern need be given to the patient's position, except as above noted, other than to avoid pressure of the neck upon the upper edge of the table. Endotracheal insufflation anesthesia has very peculiar advantages in spinal operations. As the term implies, anesthesia is maintained by ventilation of the lungs with ether vapor under a regulated pressure. The ventilation of the lungs may be effectively accomplished entirely without the assistance of the act of respiration, and indeed oxygenation of the blood is adequately maintained. Whether respiration be embarrassed by paralysis of the intercostal or abdominal muscles, as it sometimes is in spinal lesions, or by the prone position of the patient on the table, the insufflation of the lungs with air, charged with ether, by the endotracheal apparatus is quite competent not only to maintain an even anesthesia, but to oxygenate the blood adequately throughout the operation. For these reasons the use of the apparatus is strongly to be recommended not only as a matter of convenience but as a means of increasing the margin of safety.

Local anesthesia.—Under certain conditions, notably when the patient's vitality is greatly depreciated by the disease and when, as in trauma of the lower cervical cord with paralysis of the intercostal and abdominal muscles, there is the greater risk of pneumonia, I have resorted to local anesthesia. The skin and musculoaponeurotic layers are readily rendered anesthetic by the infiltration of 0.5 per cent novocain, and while it might be possible to anesthetize the periosteum of spines and laminæ, though not so effectively, when the time comes to remove these, I resort to nitrous oxid-oxygen anesthesia. Novocain infiltration of the cutaneous and muscular layers with nitrous oxid-oxygen anesthesia for the skeletal work has proven a very happy combination and may be considered an important factor in minimizing risks in desperate cases. The patient is given hypodermatically morphin sulphate 0.01 and atropin sulphate 0.0016 one-half hour before the operation; the skin along the projected incision is infiltrated with 0.5 per cent novocain solution, and after the skin flaps are reflected on either side, the aponeurosis and the muscles down to the spines and laminæ are infiltrated with massive doses of the same solution. For one operation 250 c. c. may be required. When the spines and laminæ are exposed, nitrous oxid-oxygen is given and continued until the bone work is completed. The spinal dura is more sensitive than the cranial dura and before it be incised, either it must be injected with novocain or the nitrous oxid-oxygen continued.

While this method has proved satisfactory in a number of cases, I have been impressed with the greater effectiveness of regional anesthesia, especially in operations upon the thoracic region. The sensory supply of the structures in the field of operation is derived from the cutaneous rami of the posterior primary divisions of the spinal nerves. Following the technic for alcoholic injection of the intercostal nerves, as many nerves on either side as may be necessary, according to the number of laminæ to be removed, are injected with 0.5 per cent novocain solution. It may be necessary to supplement these primary injections with injections directly in the line of incision and subperiosteally on either side of the laminæ. By a combination of these injections, I have been able to open the spinal canal for various lesions without the aid of a general anesthetic.

One or two stage operation.—The question of a one or two stage operation is often the subject of discussion, but I do not believe in an attempt to settle it, either pro or con, in an arbitrary manner. Each case is a law unto itself, but the decision rests with the judgment of the operator. I depend almost altogether upon the blood pressure as the most valuable index to the patient's condition. If the blood pressure be maintained at an approximately normal line in the preliminary stage of exposure, there is no reason whatsoever for discontinuing the operation until a second sitting. And the more attention one pays to the control of hemorrhage and the avoidance of unnecessary trauma, the less frequently will the operator be forced, as a measure of safety, to postpone the completion of the operation until a second sitting. As a matter of fact, I have had to resort to the two-stage operation but twice. The pulse rate and blood pressure are recorded on a special chart at five-minute intervals by an assistant, and a glance at the chart, constantly within view of the operator, serves to keep him accurately and continuously informed as to his patient's condition.

Instruments.—The mechanics of removing the spines and laminæ are comparatively simple, and few instruments are required. Each operator may determine for himself the instruments with which he can remove the spines and laminæ with greatest facility and with the least expenditure of time or infliction of damage. While a great variety of instruments has been suggested and recommended, I have found a modification of the Liston forceps for the removal of the spinous processes and rongeur forceps of various sizes and angles for the removal of the laminæ meet every requirement. As with all operations upon the skeleton, the bone-cutting instruments should be sharpened before every operation. Some surgeons employ saws for the laminæ, such as the Gigli saw, Horsley's saw, or Doyen's circular saw, others again the conical trephine, and while these may prove

satisfactory in the hands of those who use them, the selection of one instrument or another must be considered as matters of personal preference.

In addition to the bone-cutting instruments, but few instruments appropriate especially for spinal operations are required in the armamentarium. Among these may be mentioned two pairs of self-retaining retractors with curved teeth which are particularly helpful in that they maintain an even and constant retraction of the muscles throughout the operation and, at the same time, release an assistant's hands for other duties. Horsley's wax is required to control bleeding from the cut sections of laminæ. Mosquito forceps may be required after the dura is opened, and for cutting the theca or roots a pair of iris scissors. For the separation or isolation of roots and sometimes for retraction of the cord I have used hooks of glass instead of metal; these serve a twofold purpose. Being fragile, they serve as a constant reminder to operator or assistant of the obligation to employ the minimum amount of traction force, and as they are nonconductors, they do not transmit to an adjacent root or to the cord the electric current that we may be using for identification of individual roots. In the closure of the wound several kinds of needles should be available: For the dura, a small cutting edge, curved iris needle; for the splint sutures, a full curved Hagedorn needle; for the muscles or fascia, an Emmet needle; and for the skin a straight Hagedorn needle. The suture material includes arterial silk, iodin catgut, and silkworm gut.

Localization.—In preparation for all intraspinal operations, excluding those for trauma, but more particularly for resection of the roots, some means should be adopted for accurate localization of at least one spinous process. This is best accomplished by selecting for identification the spinous process about the middle of the contemplated laminectomy. Using the seventh cervical or vertebra prominens as a starting point in the cervicothoracic region, or the last thoracic in the thorácicolumbar region, the successive spines are counted until the desired one is reached and marked with an aniline pencil. A strip of metal is fastened to the skin at this level and a röntgenogram made. The latter will determine whether the first calculation be correct.

The operation.—With this preliminary operation, with the patient's position properly adjusted, skin disinfected, and the field of operation surrounded with sterile sheets, the operation begins. The incision is semilunar in shape, so that, upon reflection of the flap, adequate exposure is afforded of the structures beneath. The incision proportionate to the predetermined number of laminæ to be removed should be long enough to include at least one spinous process above and one below the contemplated opening, since it so often happens that one additional lamina above or below may have to be removed. This

initial incision extends to the level of the intervertebral fascia. The margins of the flap, including the skin and subcutaneous tissue, are then covered with cloths as a means of protecting the deeper structures from skin contamination.

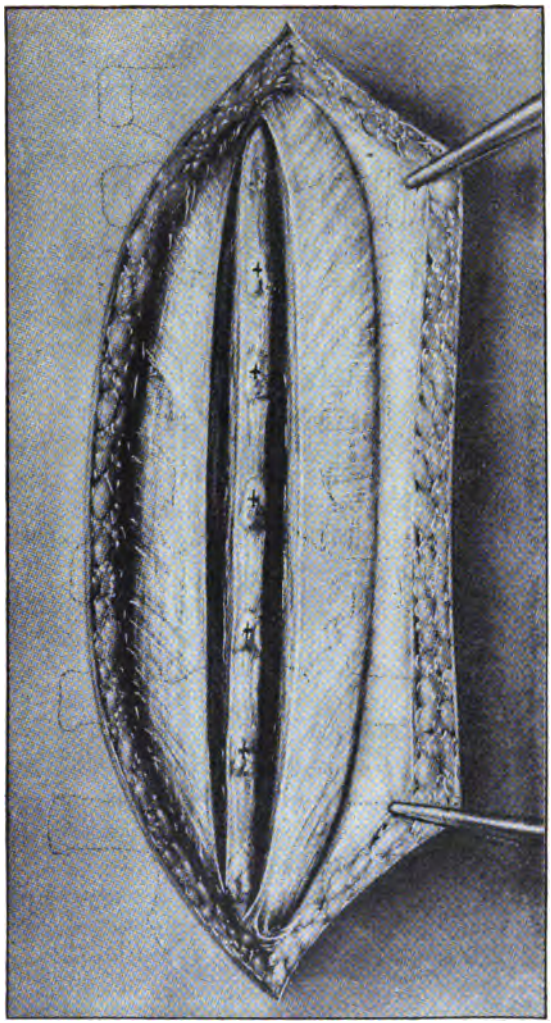

FIG. 132.—The cutaneous flap is reflected, showing the oval incision in the intervertebral fascia beginning one spinous process above and one spinous process below the contemplated opening in the spinal canal.

The incision in the intervertebral fascia begins in the median line at the tip of the spine above the first spine to be removed, and, following closely the lateral aspects of the spines, first on one, then on the other side, terminates in the median line just below the last spine to be included in the laminectomy (Fig. 132). The incision in the inter-

vertebral fascia is made with a heavy knife, and should penetrate the muscular sheath. The separation of the muscular layer from either side of the spines and from the laminæ may be done rapidly with a broad chisel. The preservation of the periosteal layer, partially at least, makes possible more or less bone regeneration, so that the defect consequent upon the removal of the spines may be in part repaired. As a matter of fact, complete bony arches, replacing the original laminæ. have been found at secondary laminectomies.

At this stage of the operation there will be more or less bleeding; more if the chisel penetrates the muscles, less if it hug closely to the spines, but hemorrhage is readily controlled by tamponing the wound at once with compresses wrung out in hot normal salt solution. Upon separation of the muscle layer on one side the wound is tamponed before proceeding with the other side, or even before the muscles have been separated from the laminæ below. Thus, by working first on one side, then on the other, complete separation of the muscles from spines and laminæ is effected with a minimum amount of bleeding and by the time this has been accomplished and the tampons removed, hemorrhage is well under control. It may be necessary to ligate several bleeding points in the belly of the muscle or in the sheaths and aponeurosis, at which level the largest blood vessels will be found. Intramuscular injections of adrenalin solution, as recommended by some, are unnecessary.

The self-retaining retractors are now introduced, one or two pairs, according to the length of the wound, and beneath them, covering the exposed muscle surface, are spread gauze pads (Fig. 133). The blades of the retractors are separated sufficiently to bring into view the articular processes on either side. Having divided with a heavy bladed knife the ligamenta interspinalis above and below, the several spinous processes are removed one by one with bone-cutting pliers and the corresponding laminæ with rongeur forceps (Fig. 134). In removal of the laminæ an opening is made with a small pair of forceps, or, as in the lumbar region, with a drill, and as soon as the space is large enough the heavy forceps are substituted, and the several arches to either side as far as the articular processes, if necessary for proper exposure, are hastily removed piecemeal (Fig. 135). While the cord is protected throughout the spinal canal by the epidural fat and in the lumbar region by the ligamenta subflava, care must be taken to avoid injuring the cord in the removal of the laminæ. This is especially true in cases of marked kyphosis, where the dura is in closer contact with the arches, and in fracture-dislocations, where the cord may be compressed by the displaced vertebræ.

Once the laminectomy is completed (Fig. 136) and before the dura is opened, the operator surveys the field in search of bleeding points.

Hemorrhage from every source should be under control before entering the dural sac, and to this end the operator will avail himself of one means or another, according to the source of bleeding. If from the bone, sterile wax is the most effective, and if from other structures, heat in the form of tampons wrung out in hot saline solu-

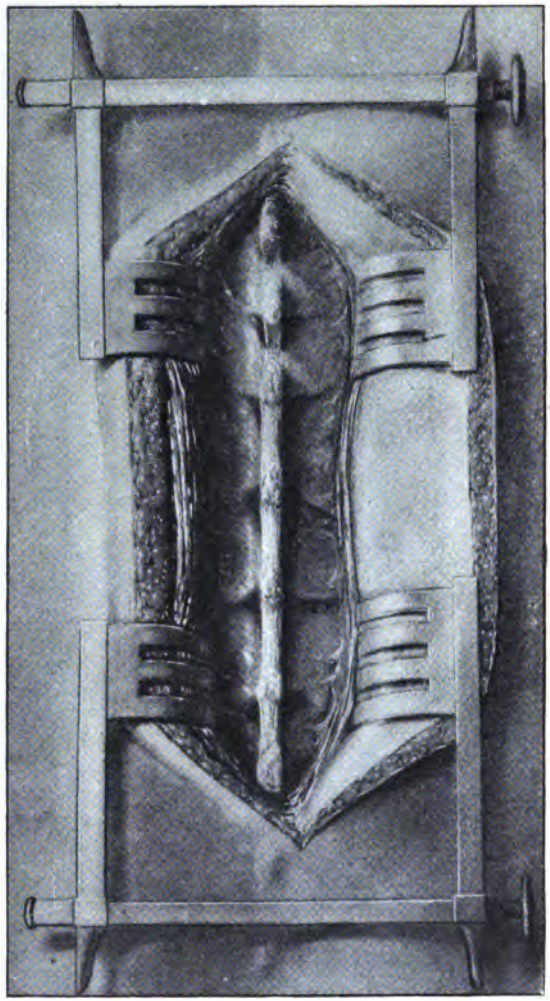

FIG. 133.—Two pairs of self-retaining retractors introduced, one at the upper and one at the lower angle of the wound.

tion, tampons of cotton or small pieces of muscle tissue will usually meet every contingency.

The subsequent steps of the operation will depend upon whe the lesion be extradural or intradural. Before opening the dur least a careful inspection should be made for extradural proce

possibly a tumor, a deformity following fracture, tuberculous foci or what not, and finding the lesion extradural, further exposure is obviated. It would be especially undesirable to invade the subarachnoid space and expose the spinal membranes or cord to infection, should the process prove to be tuberculous. A fusiform swelling of

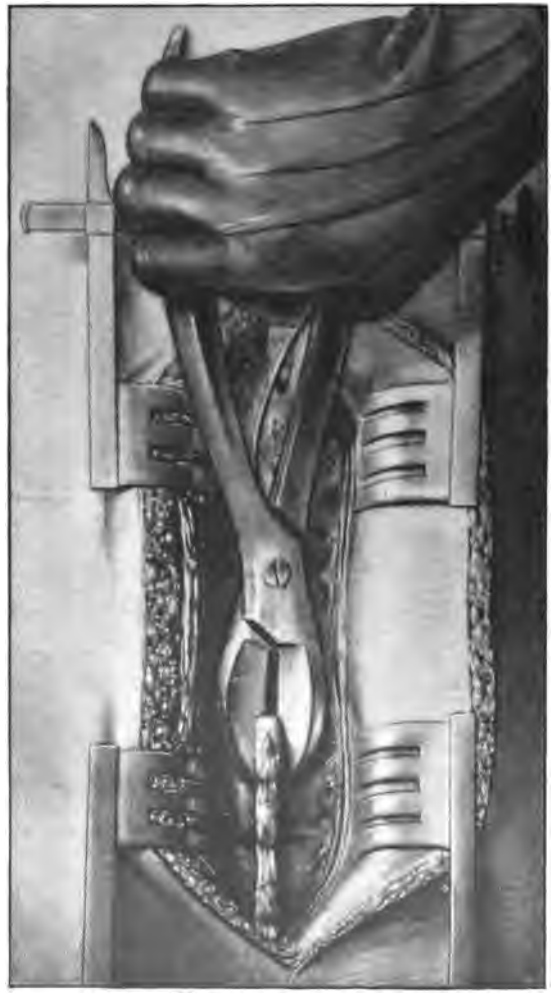

FIG. 134.—Removal of the spinous processes.

the dural sac, if present, suggests an intradural lesion and gives a clew as to its location.

Before the dura is exposed to view the thin layer of fat, together with the plexus of veins, must be displaced to either side. A careful inspection of the dura at this stage may bring to light the nature of the lesion. Under normal circumstances the dura is of a bluish-

white color and of about the thickness of the dural covering of the brain. Discoloration of the dura with or without the presence of granulation tissue bespeaks an inflammatory process. The absence of pulsation is, of course, most suggestive of a tumor, although both the arrest of pulsation and the oval swelling may be caused by a

FIG. 135.—Removal of the laminæ.

circumscribed pachymeningitis. To open the dura introduce two silk traction sutures, one on either side, mounted on small curved needles, which enter but do not penetrate. Slight traction upon these sutures frees the dura, and between these a minute incision is made with a small scalpel down to but not including the arachnoid. The latter lies in such close apposition to the dura that in some cases

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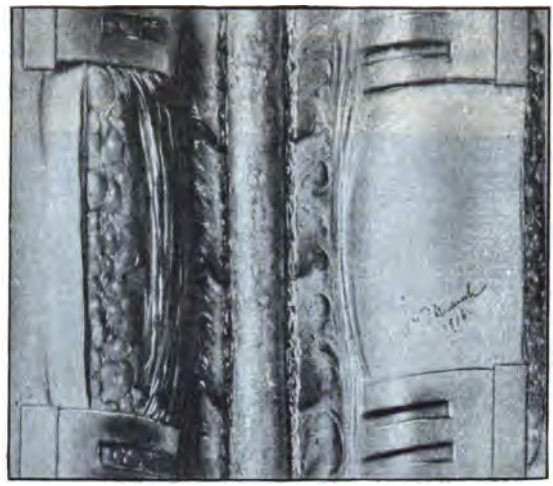

FIG. 136.—Exposure of the spinal cord after the removal of the laminæ.

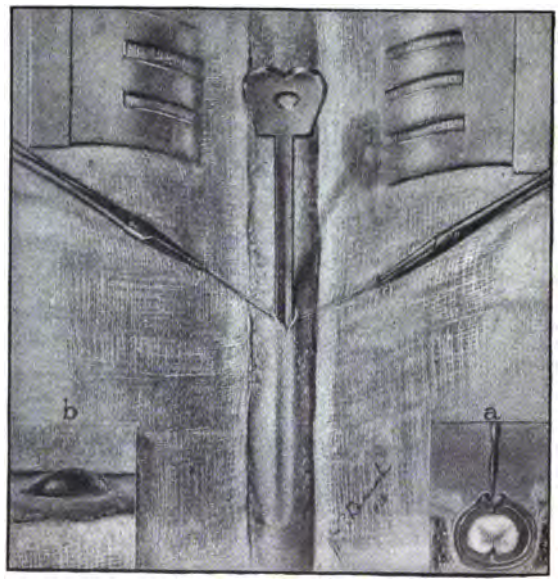

FIG. 137.—The introduction of the groove director through the initial opening in the dura. *a*, The knife in place in the groove of the director as the dural incision is made; *b*, the thin filmy arachnoid membrane ballooning through the dural incision.

even with care the scalpel will puncture it and the cerebrospinal fluid gush forth. But if not perforated, the arachnoid will balloon through the dural incision under the pressure of the pent-up fluid. The dural incision is lengthened by cutting over a director first in one direction, then in another (Fig. 137). Before incising the arachnoid, four additional traction sutures are introduced through the margin of the dura—two above and two below those already in place. The three pairs of traction sutures are grasped separately with mosquito for-

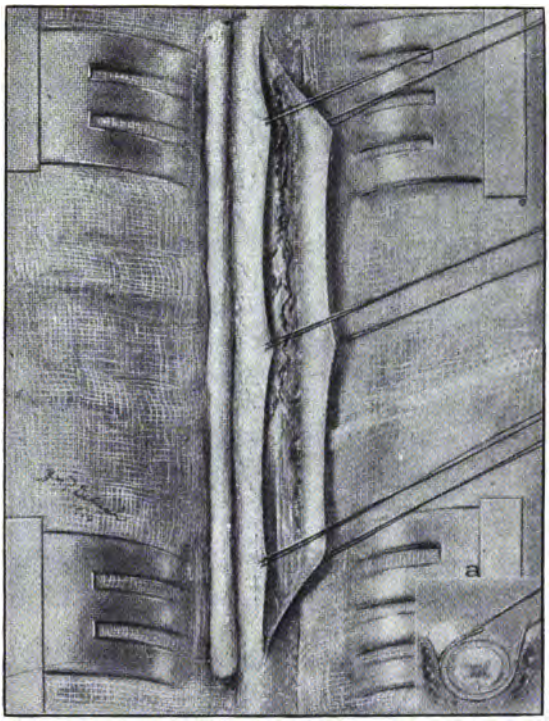

FIG. 138.—After the three pairs of traction sutures have been introduced, a roll of cotton is laid in the trough on either side of the dural flap to absorb the blood, which may gravitate to the bottom of the wound, and to prevent its entrance within the dural sac. *a*, Representing in cross section the relation of the cotton roll to the dural sac.

ceps, which by their weight alone will retract the dural flaps sufficiently to afford ample inspection of the subarachnoid space. But before retracting the flaps a roll of cotton a centimeter in diameter and the length of the dural incision is laid in the bed of the wound to the outer side of each dural flap (Fig. 138). These cotton rolls will absorb the blood that may gravitate to the bottom of the wound during the operation and prevent its entering the subarachnoid space. If at any time they become saturated, fresh ones should be substituted.

While perfect hemostasis is unquestionably a *desideratum*, in the strictest interpretation this is not feasible, and yet it is entirely possible to prevent leakage into the subarachnoid space. For this reason the cotton roll plays a very important rôle.

The operator pauses now to inspect the field, and with the arachnoid still intact, his observations may give a clew to the seat and character of the lesion. Under normal circumstances the cerebrospinal fluid pulsates synchronously with the pulse and with respiration. In many instances the respiratory movements may be seen when the pulsations are not visible. This natural phenomenon may not be present if the subarachnoid space above the opening be so obstructed that free communication with the subarachnoid space above is interrupted. In addition to the presence or absence of pulsation, the operator informs himself as to the degree of tension of the cerebrospinal fluid, and as to whether the cerebrospinal fluid be excessive; he notes whether the excess of fluid be evenly distributed or circumscribed as in circumscribed serous meningitis. In the case of intradural tumor an excess of fluid is found usually above the tumor, though occasionally below, but in either case the fluid may be under such tension that not the slightest pulsation can be detected. Once the subarachnoid space is open the cerebrospinal fluid escapes under varying degrees of tension. Through the initial puncture of the arachnoid I have seen the fluid spurt into the operator's face.

The importance of hemostasis and its means of accomplishment have been discussed, so that it suffices at this time to call attention to them merely by way of emphasis. The second important injunction, the avoidance of trauma, applies, of course, only to the steps of the operation that have to do with the spinal membranes, the roots, and the cord.

I have been convinced of the evil effects of trauma by many operative experiences, particularly those upon the roots. Not until it becomes necessary to manipulate the roots, as in separating the anterior from the posterior, will any material change be noted on the pulse or blood pressure. The application of a pledget of cotton, saturated with 1 c. c. of 0.4 per cent stovain, to the cord and roots at the site of manipulation or just above it has a positive inhibitive influence.

While I do not believe the sudden escape of cerebrospinal fluid is the cause of spinal shock, I can readily see how undesirable it would be to allow the dural sac, ventricles, and cisterns to be emptied of their fluid contents during operation. The continuous flow of fluid is prevented partly by arching the patient's back so that the opening in the dura is at the highest level of the subarachnoid space, but to keep the field entirely dry, posture must be supplemented with small tampons of cotton gently introduced between cord and dura just

above and below the upper and lower limits of the dural incision. With this preparation the operator is prepared to inspect the meninges, roots, and cord, and the subsequent steps of the operation will depend upon the nature of the lesion.

Closure of the wound.—Before closing the dural wound hemorrhage must be under absolute control. The presence of even a small amount of blood with its fibrin content leads inevitably to the development of adhesions, and though this may not lead to serious consequences, every effort should be made to avoid them and to leave the structures within the sac as nearly free from the effects of trauma as possible. But a freer hemorrhage within the sac is responsible unquestionably for some of the functional disturbances, either transitory or permanent, that have followed laminectomies. The presence of complete or partial paralysis of one or both extremities, or of bladder or rectum, may be the result of trauma incidental to traction upon the cord or roots or possibly to the resulting edema, following exploratory laminectomies, but the persistence of these motor phenomena should be charged in most instances to hemorrhage. Perfect hemostasis is, therefore, a sine qua non in preparation for closure of the dura. There are occasions when with propriety the dural incision may be left unsutured. Pachymeningitis hypertrophica cervicalis chronica and inoperable tumors are conditions in which for the relief of pressure it is advisable not to suture the dura, and in meningitis serosa chronica there are some who believe in the advantages of providing for drainage, thus preventing the reaccumulation of cerebrospinal fluid. With this view, however, I am not in accord.

To close the dural incision a small, curved iris needle and 00 silk should be used. A continuous suture is preferable to an interrupted suture as a safeguard against leakage and the establishment of a cerebrospinal fistula (Fig. 139). The needle is introduced precisely in the edge of the dural incision, the suture is continuous, and with the wound thus closed, no foreign material will be exposed within the dural sac and there can be no leakage through the needle punctures and no leakage along the line of suture. Observing these details in the technic of dural suture, I have never seen any escape of cerebrospinal fluid after laminectomies.

To obliterate the " dead space " that remains after removal of the spinous processes and laminae, three or four splint sutures, according to the length of the incision, are introduced, and after closure of all the layers of the wound is completed, one end of the suture is threaded through a section of narrow rubber tubing so that when tied the suture will not cut through the skin. The next row of sutures of continuous iodin catgut brings the sheath and margins of the erector spinales into apposition, and over this the intervertebral fascia is brought into apposition with interrupted sutures of catgut

(Fig. 140). The intervertebral fascia is a strong unyielding structure and unquestionably plays an important part in preventing such incapacity as might come from the removal of the spines and interspinal ligaments. As giving support to the structures of the back after laminectomies, it plays a part analogous to that of the fascia of the external oblique after operations in the lower abdomen. There remain to be closed the skin and the superficial fascia, which are brought into apposition separately, the latter, with interrupted catgut sutures and the former with sutures of interrupted silkworm gut. Finally, the splint sutures are tied, the skin again disinfected with iodin and the dressing applied.

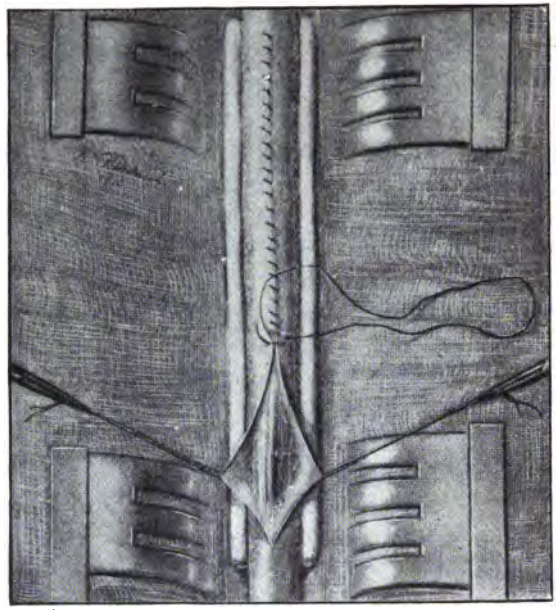

FIG. 139.—Closure of the dural incision with continuous silk sutures.

After the operation.—The management of the patient after a laminectomy must take into consideration various conditions that do not pertain to operations elsewhere. With decubitus already present, or from the nature of the lesion likely to develop, the patient is placed upon a water or air mattress. On the bed immediately beneath the dressing I place a square of wax paper covered with sterile cotton to absorb any blood that may ooze through. This is merely a precautionary measure. I have never had occasion to use fixation dressings, either of plaster of Paris or poroplastic felt. There is no indication for fixation, except perhaps in the cervical region, where the use of small sand bags on either side of the head and a strip of adhesive plaster from one to the other, crossing the patient's forehead, meets every need.

When the operation has been in the lumbar region the nurses and attendants should be warned of the risks of contamination of the dressings with the excreta, and as a protection the dressing should be hermetically sealed around the edges as well as over the surface with adhesive plaster. With an incontinent bladder either a suprapubic drain or a permanent catheter is installed, and retained at least until

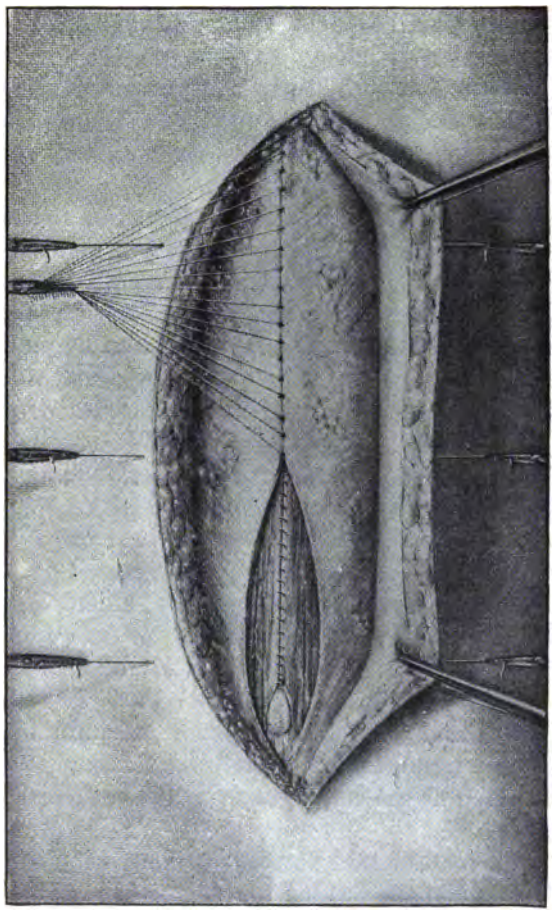

FIG. 140.—Interrupted catgut sutures in the intervertebral fascia.

the wound is healed. The wound itself requires little attention; save for the removal of a drain at the end of twenty-four hours, the dressing is not disturbed until the seventh day for the removal of stitches, at which time a protective pad is applied and allowed to remain until no further protection is required.

The escape of cerebrospinal fluid after laminectomies is more or less of a hoax. When the dural wound is closed, as I have directed, with a continuous silk suture properly introduced, no cerebrospinal

fluid will escape, and I might even go so far as to say that even when I have had to remove a portion of the dura I have had no apprehension of a cerebrospinal fistula, since the closure of the musculocutaneous wound with four tier sutures, one each in the muscles, aponeurosis, superficial fascia and skin, is an absolute guarantee against leakage. When cerebrospinal fistulæ have been established there is, of course, the danger of infection, and the convalescence will be embarrassed with intense headache and vomiting, sweating, hyperpyrexia and acceleration of pulse. In some cases it is reported the fistula closed spontaneously, in some the patient died of meningitis. To arrest the flow and to favor spontaneous closure, the foot of the bed should be elevated and, as a guard against infection, the wound should be cared for most scrupulously.

The condition of the patient after the operation in many instances is not such as to give alarm. The pulse may be accelerated, the temperature subnormal, and the skin leaky, but it is unusual to see the patient in a condition even of surgical shock or collapse. In the majority of cases, therefore, with the exception of morphin for the relief of pain, little or no medication is required, and because the sphincters are frequently paralyzed and because the dressing may become soiled by leakage, proctoclysis is not to be employed.

The most constant and often the only subjective disturbance is pain. Whatever may be the experience of others, I find that most of my patients have enough pain to require one or more hypodermics of morphin during the first 24 hours. The degree of pain will depend somewhat upon the nature of the operation, chiefly upon incidental trauma of the roots; so that after rhizotomy or the disentangling of a tumor from the roots, pain will be intense and morphin or codein is given just as liberally and frequently as may be necessary to insure the patient's comfort. The effect of morphin upon the pulse rate under these circumstances is striking, so that morphin is just as clearly indicated as the employment of general cardiac stimulants are uncalled for.

No general rule can be laid down as to the period of enforced rest in bed. The extent of the laminectomy, whether in the cervical, thoracic, or lumbar region, must be taken into account. In a limited resection, as of three or four cervical or thoracic vertebræ, I allow the patient to sit up at the end of the second week, but after more extensive laminectomies, especially in the lower thoracic and thoracicolumbar regions, the patent is content to remain in bed two weeks longer. In so many instances the patient is already paralyzed before the operation that the question of his becoming ambulant depends more upon the paralysis than upon how many laminæ were removed.

Complications.—The complications peculiar to laminectomy are chiefly those which arise from intentional or unintentional, avoidable

or unavoidable, damage to the cord and roots; that is, motor or sensory disturbance, transitory or permanent. In the removal of tumors, the displacement of the cord, the separation of roots, involves a degree of trauma that may give rise to temporary disturbances of function, more often motor than sensory. Thus, there may be complete or partial paralysis of one or both lower limbs and of bladder and rectum, abdominal distention and anesthesia below the level of the operation. Although distention is a more frequent complication of operation upon the lower thoracic vertebræ, the abdomen becomes distended, sometimes to such a degree as to cause more or less respiratory embarrassment from pressure of the distended bowel upon the diaphragm. When, coupled with this, peristalsis is arrested and the patient is unable to pass gas, the condition presents a picture not unlike that of peritonitis minus tenderness. Fortunately, the distention is of short duration, disappearing gradually in two or three days, and unless the patient be in distress the condition may be allowed to pass untreated. In the more exaggerated forms I have found the use of the rectal tube and 1 c. c. of pituitrin hypodermatically offered the greatest measure of relief.

After laminectomies there may be either retention or incontinence. Retention of urine is really quite common, irrespective of what may have been the condition of the bladder before the operation, and restoration of bladder function may be a matter of days or weeks. When of short duration, intermittent rather than permanent catheterization is preferable, but if after a few days there is no tendency to immediate restoration of function, permanent catheterization can not be avoided. One can not always rely upon the patient's sensations, as in certain regions, especially the lumbosacral, the patient is unconscious of the sense of overdistention. Incontinence of urine is a very much more grave complication and implies more serious damage to cord or meninges. Whether it will be permanent or transitory may usually be determined by the nature and seat of the lesion. I have seen a number of cases of transitory retention, but only one instance of permanent incontinence.

Paralysis of bladder, rectum, or extremities, developing as complications after operations within the dural sac, wholly irrespective of what might have been due to the lesion, are unquestionably the expression of pressure within or without the cord, and whether they be transitory or permanent depends altogether upon the nature of the cause. It has been said by some that an excessive collection of cerebrospinal fluid may be held responsible for the pressure symptoms. This seems to me possible but unlikely. I believe the most common cause is hemorrhage without the cord into the subarachnoid space or hemorrhage and edema within the cord, and depending upon the extent, but more especially upon the seat, of the hemorrhage, are the

symptoms transitory or permanent. The effects of a moderate hemorrhage without the cord into the meshes of the arachnoid would hardly be more than temporary; the same might be said of a transitory edema within the cord. But hemorrhage within the cord may readily lead to permanent structural changes so extensive as to cause a complete transverse lesion. The importance, therefore, of the avoidance of trauma and strict hemostasis is again emphasized.

Pneumonia or hypostatic congestion is a complication of cord lesions rather than of laminectomy, *per se*, but it is mentioned at this time merely to suggest the propriety of elevating the patient's head and trunk as a prophylactic measure. This applies only to cases with paralysis of the respiratory muscles, as in cervicothoracic lesions.

The prevention of adhesions is a pertinent question. If for any reason the necessity of reopening the dural canal arise, the procedure will be devoid of difficulty, providing there are no intramembranous adhesions, and the converse of this may be stated in more forceful terms. The presence of adhesions is a very grievous obstacle to exploration. How to prevent them is, therefore, a vital question. To deny the tendancy for them to form is to close one's eyes to the truth, and to account for their existence when reasonable precautions are taken is somewhat baffling. I have given much time and thought to this problem, and, excluding infection, the possible causes of adhesions to my mind may be narrowed down to hemorrhage and trauma. As for hemorrhage the deposition of the fibrin in the blood between the membranes in the course of the operation provides at once a means whereby a point or area of adhesion may be formed. On this account particularly must minute care be taken to protect the subdural sac from contamination with blood, and to this end bleeding should be controlled wherever possible before the dural sac be opened. But there will be more or less uncontrollable oozing to be provided for, and to protect the sac from this I have suggested the use of tampons of cotton laid one on either side of the dural incision, so that when the dural flaps are reflected with traction sutures, the cotton tampons will prevent the overflow of blood into the dural sac. Thus, it is a comparatively simple matter to prevent the deposition of fibrin within the sac when the source of hemorrhage is extradural. Not so, however, when the hemorrhage is subdural, since the necessities of the situation, the removal of tumors, the cutting of roots, will provoke more or less bleeding, the control of which will tax the resources of the operator.

The second source of adhesions is the exudate that may form in the membranes as a result of trauma or in the process of repair of the dural incision. The endothelial lining of the dura, like that of intima and peritoneum, is so sensitive to the insult of trauma that it demands the greatest respect.

The laminectomized spine.—There has been, and still is, a very prevalent belief—not among surgeons, however—that the removal of spines and laminæ robs the patient of the ability to support the weight of the head or body in greater or less measure, and that after laminectomy the strength of the back is seriously impaired. This is an entire misconception of the facts. Necessary as the spines, laminæ or intraspinal laminæ might seem to be, it is surprising how little the removal of these structures interferes with the carriage of the body at rest and in motion. This general statement must be qualified in two particulars, first, when the structure of the vertebral bodies are tuberculous or the seat of a malignant tumor, the removal of spines and laminæ may be a matter of some consequence and call for some methods of artificial support. It has been suggested that under such circumstances the spinous processes, laminæ, and articular processes above and below the defect may be lashed together with strong silver wire. I have never had occasion to adopt this suggestion, practical as it seems, and would hesitate to do so because of the possible complications of wound repair. Theoretically, the demands for this method of giving greater security to the spine are not very great; if the indication be of malignant disease of the vertebral bodies, a spinal brace would answer the purpose quite as well for the time that remains until the patient becomes bedridden, and if the indication be of tuberculous spondylitis, the lesion itself would necessitate the use of a brace or some other fixation appliance. Secondly, the restoration of function in the laminectomized spine will be influenced, not by the number of spines removed but by their location. The only disability that has been brought to my attention was after the removal of the laminæ of five lumbar vertebræ. One of my patients, a middle-aged man, suffered more or less discomfort in the form of pain on certain movements and a feeling of lack of support. Naturally there is more strain at the lumbosacral junction than elsewhere, not only because it is at the base of the spine and supports the entire body but because this is the junction between a movable and a fixed point in the vertebral column, a point which of necessity is subjected to greater strain.

Leaving out of consideration, therefore, laminectomies at the lumbrosacral junction and in the presence of softened vertebral bodies, we now have no hesitation in removing as many consecutive spines and laminæ as may be required for thorough exploration or for adequate dealing with the lesion itself. The osteoplastic flap is not essential to the maintenance of function, but perfect apposition of the several musculo-aponeurotic layers in the closure of the wound plays a very important part.

CHAPTER IX.

PERIPHERAL NERVES.

In the course of the present war, nerve injuries have formed a very considerable percentage of the total casualties. According to Tinel, nerve trunks are injured in at least 18 to 20 per cent of wounds of the extremities. Evidence of nerve injury must therefore be sought for in all such wounds.

As the result of a wound, the nerve trunk may be injured in a variety of ways. It may be *completely cut through* by a shell fragment; it may be *compressed* primarily by fractured bone or bullet, or secondarily by scar tissue, callus, etc. *Bruising* of the nerve may lead to hemorrhage within the sheath and a secondary fibrosis which may convert the entire cross section of the nerve into a fibrous band. Or the nerve may be involved in an area of local ischemia, as in Volkmann's paralysis.

It is often impossible to determine from our examination the exact pathological condition of an injured nerve. We are therefore compelled to use a clinical classification, and that classification is based on whether or not that nerve is capable of conducting impulses. If a nerve has completely lost the power of conducting impulses, we speak of "*complete division.*" If it has only partially lost the power of conducting impulses, we speak of "*incomplete division.*" Complete division in this sense does not necessarily mean that the nerve is cut in two. A nerve whose physical continuity is preserved may, for example, as a result of a complete fibrosis of its cross section, be unable to conduct impulses. Therefore we further divide each group into anatomical—where there is a physical break in continuity—and physiological—where there is no anatomical break. Nerve injuries then are divided as follows:

1. Complete division: Anatomical; Physiological.
2. Incomplete division: Anatomical; Physiological.

If, then, our clinical examination shows that a nerve fails to conduct any impulses, we speak of complete division of that nerve, without committing ourselves as to whether the loss of conductivity is due to an anatomical or physiological break, or, as often happens, to a combination of the two, e. g., section of half the nerve, with compression of the rest of the nerve by scar tissue.

Similarly, when we speak of incomplete division, we mean that the conductivity of that nerve is impaired, whether as a result of cutting, crushing, pressure, or fibrosis. In an incomplete division the damage may be confined to one part of the cross section of the nerve, leaving the rest intact, or it may affect the whole cross section of the nerve more or less diffusely. Therefore, our symptoms of incomplete division will vary not only with the extent of damage done but also with its distribution.

It is a fundamental principle that every nerve fiber, when separated from its cell of origin, degenerates. This degeneration, the

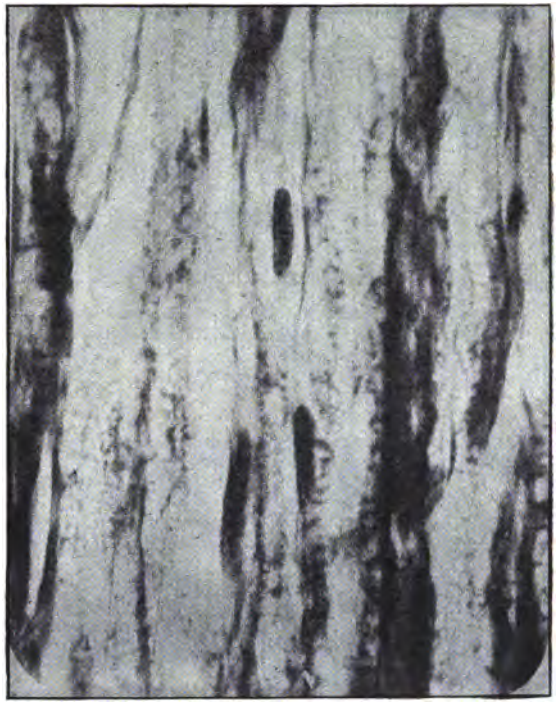

FIG. 141.—Normal nerve indicating the maximum number of neurilemmal nuclei. One nucleus is found normally in each internodal segment which measures from 80 to 1,000 μ. (Kirk and Lewis.)

so-called Wallerian or secondary degeneration, consists in fibrillation and destruction of the axis-cylinders, breaking up and disappearance of the myelin, and, finally, proliferation of the cells of the sheath of Schwann. After degeneration has taken place, the nerve fiber exists as a thickened neurilemmal sheath containing a nucleated protoplasmic band; and so it remains for a long time, ready to receive the down growing axis-cylinders as soon as regeneration commences. The existence of such a nerve sheath acts in some way not clearly understood to attract and direct the down growing fibers.

FIG. 142.—Preparation from the distal segment of a nerve six days after division showing active hyperplasia of neurilemmal nuclei—and beginning formation of protoplasmic bands. (Kirk and Lewis.)

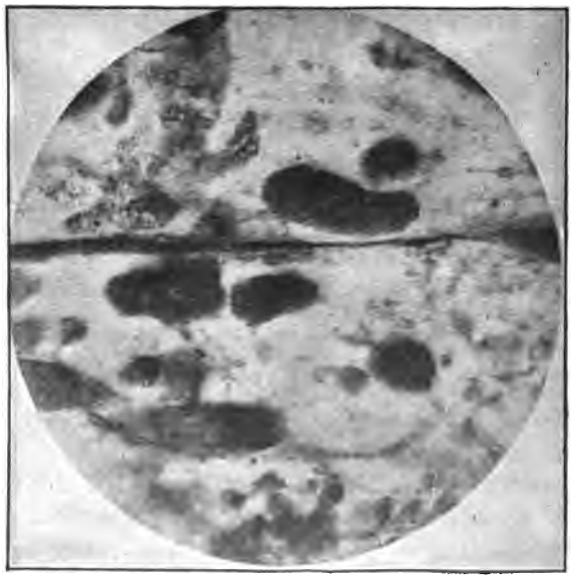

FIG. 143.—Protoplasmic band formed from nuclei of neurilemmal sheath containing newly formed axis-cylinder. (Kirk and Lewis.)

If, then, a nerve is severed and the end separated from its cell, it degenerates. If the distance between the severed ends is not too great, and no serious obstruction exists, the nerve fibers grow down along the old nerve tract; and as they grow, they are guided apparently by the cells of Schwann, and there is a gradual reconstruction of the degenerated nerve. Under favorable circumstances complete regeneration with complete return of function occurs.

If the gap between the severed ends is too wide, or the scar tissue too dense, regeneration is impossible. In such cases, the new form-

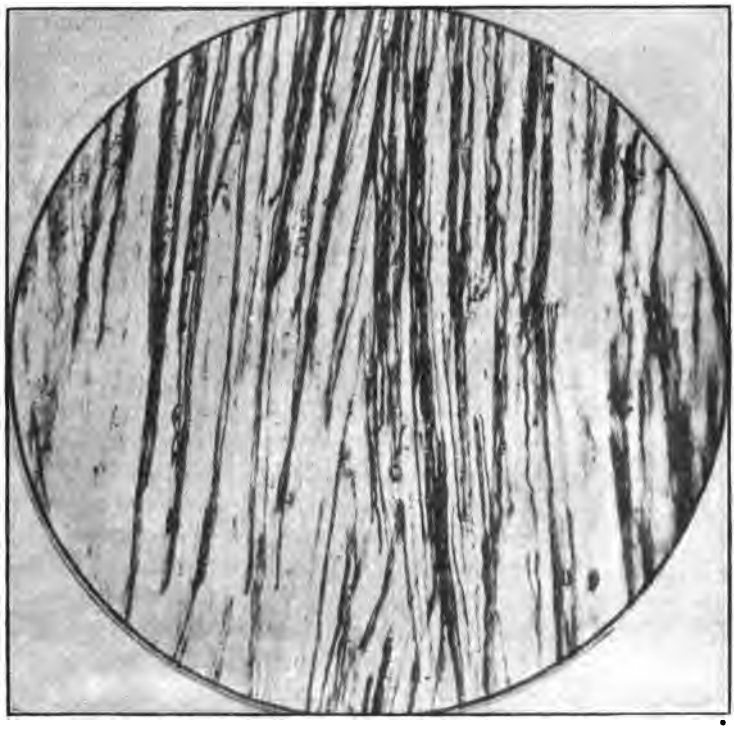

FIG. 144.—Newly formed neurofibrillae with terminal end bulbs. Large numbers of neurofibrillae develop from these end bulbs. (Kirk and Lewis.)

ing nerve fibers will either stray about in the neighboring scar tissue, or mat together at the level of the lesion, along with proliferated Schwann cells and connective tissue, to form a bulbous neuroma, e. g., amputation neuroma.

Where severe damage to a nerve occurs, without separation of the segments, a bulbous swelling appears at the site of the lesion, composed of regenerating neurofibrils, scar tissue, and Schwann cells in a complex mass (Fig. 145). A lesion such as here represented may form a complete obstacle to the downgrowth of new fibrils, and as such render spontaneous regeneration impossible. But in similar

lesions of slighter degree with less scar tissue formation, a variable amount of regeneration will be possible, and finally we reach a stage where, only a very small amount of damage having been done to the nerve, complete restoration to normal can occur.

In all these cases we have assumed an injury severe enough to cause degeneration of the nerve distal to the lesion. Even if complete recovery takes place, it will be long delayed. Under favorable conditions, nerve fibers may grow 1 to 2 mm. a day, and at this rate of growth considerable time must elapse before the nerve reaches its peripheral end organ and function returns.

FIG. 145.—Total interruption. Neuroma, above; pseudo-neuroma, below. (Tinel.)

With very slight damage to the nerve, such, for instance, as caused by slight compression, we may get an interference with nerve function (some loss of sensation and motion) without sufficient damage to the fibers to cause them to degenerate peripherally. In such a case, relief of pressure would be followed by prompt recovery.

History and examination.—It is not until some time after a nerve injury that the signs by which one can clearly recognize the nature and extent of the lesion develop. Ten days to two or three weeks must elapse before the first stage of the picture is clear. From then on repeated examinations may be necessary to determine the exact location and degree of the trouble.

History: Date of wound; nature of projectile; position of limb when struck; other injuries suffered at the same time; loss of blood; use of tourniquet or tight bandage, and how long applied; fracture; infection; surgical intervention (ligation of arteries, suturing nerves, etc.).

Paralysis: Developed at time of wound or later; course of development.

Paresthesias: Numbness, tingling, pain. Time of onset and course of development.

Vasomotor disturbances: Time of onset and course of development.

Treatment: X-ray.

Examination.—A. General.

(*a*) Joints. Freedom of movement. Extent of ankylosis.

(*b*) Bones. Evidence of fracture. Callus.

(*c*) Wounds and scars. The course of the projectile should be determined if possible.

(*d*) Position of injured part, or limb. Faulty position may be due to fractures, deformities, joint lesions, nerve lesions, or nerve lesions with secondary contractures. Where a motor nerve is cut the muscles supplied are paralyzed. The antagonistic group of muscles overact, gradually stretching the paralyzed muscles. At first this

FIG. 146.—Complex neuroma. Above, penetration of the central end, the majority of whose fibers have undergone fibrillary transformation. Some fibers are rolled in spiral form above the neuroma. In the middle part, neuroma made up of the intercrossing and grouping of axis-cylinder fasciculi (fibers sectioned longitudinally or transversely). In the lower part of the neuroma is found a denser fibrous tissue which probably represents the cicatrix of the wound. Below, the cells of the empty sheaths form cellular columns, the proliferation of which forms on the left a veritable pseudo-neuroma. On the right, a few regenerated fibers have succeeded in passing into the peripheral (semi-diagrammatic) segment. (Tinel.)

position is readily reducible, but eventually may become fixed by permanent shortening of the antagonistic group.

B. Motor phenomena.

Following the section of motor nerve fibers that supply a muscle there develop: (*a*) Paralysis of the muscle; (*b*) loss of muscle tone

(hypotonia, atonia); (*c*) loss of reflexes; (*d*) atrophy; (*e*) changes in the irritability of the muscle.

(*a*) In testing voluntary power (see table of muscle action, Table V) it is not sufficient to test each movement, as movements are generally complex and carried out by a group of muscles. Each muscle taking part in a given movement must be separately examined, and the degree of weakness determined. If great weakness exists, the limb must be placed in such a position that the force of gravity will be eliminated. For example, in testing for extension of the wrist the arm should be placed in a position midway between pronation and supination. Where the limb fails to move, one still may be able to feel a contraction of the tendons.

3

FIG. 147.—Pseudo-neuroma resulting from bruising without interruption of nerve fibers. Rarefaction and fibrillation of axis-cylinders. (Tinel.)

(*b*) Normal muscle tone is dependent on the integrity of the reflex arc, which consists of afferent fibers from the muscle to the spinal cord, and efferent fibers from the spinal cord to the muscle. When this arc is broken at any point, loss of muscle tone results. This may be recognized by the loss of normal contour, flabbiness to touch, and lack of resistance to stretching.

(*c*) The deep reflexes, or tendon jerks (a list of which will be found in Table VI) are likewise dependent on the maintenance of muscle tone. Diminution of tone results in the lessening or abolition of the reflex.

(*d*) Following section of a motor nerve, the muscles supplied undergo atrophy. The atrophy appears within two or three weeks, and gradually becomes more pronounced. It is recognizable by diminution in size of the muscle and its soft and structureless consistence.

Along with the wasting of the muscle fibers, there may be a progressive proliferation of connective tissue. If the atrophy is marked, and degeneration does not soon occur, the muscle may undergo complete fibrosis. Keeping up the circulation of a paralyzed muscle by massage, etc., will help to prevent this fibrosis.

(*e*) Idio-muscular contraction: Percussion over a normal muscle produces a contraction of the muscle fibers struck with the formation of a local welt. This is the mechanical muscle contractility, or idiomuscular contraction. It has no relation to muscle tone. Idiomuscular contractility is *increased* in peripheral nerve lesions.

The extent to which these various motor phenomena develop, depends on the degree of nerve injury. In case of complete nerve section, there is complete paralysis of all the muscles supplied, complete atonia, loss of reflexes, and marked atrophy.

If the lesion is incomplete, the extent of the motor phenomena is proportional to the degree of the lesion. The muscles are weak, but not completely paralyzed. Some may escape damage altogether. Muscle tone is reduced (hypotonia) but not lost. There is reduction or loss of reflexes, and a variable degree of muscle atrophy.

C. Sensation.

In discussing the sensory phenomena we must review briefly the work of Head and his colleagues. These workers divide sensibility into three main types—(1) Epiciritic; (2) protopathic; (3) deep.

Epicritic sensibility includes the recognition of *light touch* (cotton), minor differences in temperature, recognition of compass points, and accurate localization of the spot touched.

Protopathic sensibility includes the recognition of cutaneous pain (pin prick), and major difference in temperature.

Deep sensibility includes the recognition of pressure (deep touch), pressure pain, and muscle sense (sense of position and passive motion).

The fibers subserving deep sensibility run to the muscles along with the motor nerves. They have very extensive anastomosis, and so in general, in nerve injuries, the loss of deep sensibility is much less extensive than the loss of cutaneous (epicritic and protopathic) sensibility.

For this reason it is necessary to exercise care in testing sensation. If instead we use firm pressure, e. g., with a pencil, instead of light touch (with cotton), we may miss the loss of cutaneous sensation over an area where deep sensibility is retained.

After the section of a cutaneous nerve, we find a loss of sensation corresponding fairly well to what we recognize as the anatomical distribution of that nerve, as pictured in text books of anatomy. This loss of sensation, which we map out by testing with cotton, is the epicritic loss.

Lying within this zone, and smaller than it, is an area over which sensation of pain on pin prick is lost. This is the area of protopathic loss. (Fig. 148.) Between the zone of epicritic loss and the zone of epicritic plus protopathic loss (shaded area) is an intermediate zone. In this area light touch is lost; pin prick is felt as pain; but the pain is not sharply localized, it has an unpleasant, tingling, radiating character.

The peripheral nerve is the unit of epicritic sensibility. When a peripheral nerve is cut, the loss of epicritic sensibility is generally

greater than the loss of protopathic. This is because the protopathic fibers possess wider anastomosis.

On the other hand, the posterior root is the unit of protopathic sensibility. And following section of a posterior root, the loss of protopathic sensibility may be equal to, or greater than that for epicritic.

Testing sensation.—If pain is complained of, it should be determined whether there is any tenderness of the skin. Its extent may be marked out by dragging the point of a pin across from the sound to the affected side.

The test for epicritic sensibility should be made with cotton, or a light camel's-hair brush. The presence of hair may interfere, as stimulation of hairs gives protopathic and not epicritic sensibility. For accurate work it may be necessary to shave the part.

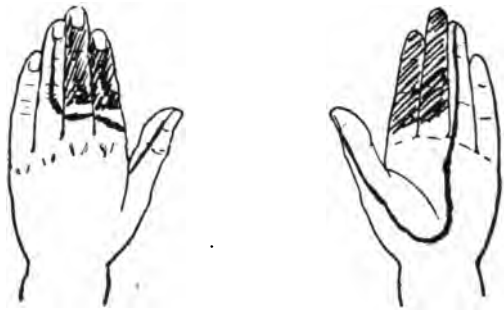

FIG. 148.—To illustrate the loss of sensibility resulting from division of a peripheral nerve. The area of loss of sensibility to light touch is bounded by a line; the shaded portion represents the area of loss of sensibility to prick and all degrees of temperature. The unshaded portion represents the "intermediate zone."

For protopathic tests, a sharp needle or pin should be used. The patient must respond only when he feels the pain of the stick (protopathic). If he feels the pressure of the pin, but not the pain, protopathic sense is lost, but pressure (deep) sense retained.

For testing deep sensibility we may use pressure of a pencil or blunt object.

Repeated sensory examinations may be necessary, and the results should always be charted for purposes of comparison.

Complete division of a peripheral nerve results in complete and persistent loss of all forms of sensation carried by that nerve. For the ordinary peripheral nerve, this will mean a loss of epicritic sensibility over a region, different for each nerve, the boundaries of which are fairly constant, and are represented in chart (peripheral nerve chart). The loss of protopathic sensibility will generally be smaller (as explained above). It is more variable from patient to

patient. The loss of deep sensibility will be the smallest of all. Cutting of tendons will considerably increase the zone of loss of deep sensibility.

Where a nerve is incompletely divided, conditions are different. It was formerly thought that in a mixed nerve, the motor fibers are much more sensitive to injury than sensory fibers. Recent work of Sherren has made it appear that the reverse is true; and that if an injury is sufficient to damage a nerve, it will damage the sensory fibers first. If the injury is not severe, there will be loss of epicritic alone, with retention of protopathic. In the slightest degrees of damage, the epicritic sensibility is not lost, but the patient is conscious of a "difference of feeling," as the cotton is dragged across the affected area. With greater damage, protopathic loss also occurs. In partial nerve lesions of this type, it is not necessary that the sensory loss includes the entire sensory distribution of the nerve. The zone of sensory loss may be smaller than that occurring in complete division of the same nerve. Disturbances of this kind, such as epicritic loss with retention of protopathic sensibility, or an incomplete loss of both, is in itself strong evidence in favor of an incomplete as distinguished from a complete nerve lesion.

Electrical diagnosis.—Among the symptoms that are of service in determining the severity and location of a lesion of the peripheral motor neuron, i. e., either the cells constituting the nuclei of the motor cranial nerves, those in the anterior horns of the gray matter of the spinal cord, or the nerve fibers coming from them, is the peculiar way in which these nerves and the muscles supplied by them behave when stimulated with electricity. The faradic or induced, the galvanic or constant currents and discharges from a condensor may be used for this purpose. The faradic and galvanic currents are those most commonly employed.

The changes which then occur are known collectively as the *reaction of degeneration* or, as often abbreviated, the *DeR* or *DR*. This has been defined by Erb to be an entire cycle of quantitative-qualitative changes of irritability, which occurs in the nerves and muscles under certain pathological conditions and presents intimate relations to certain histological degenerative changes occurring in these structures. It is characterized, in the main, by diminution and loss of the faradic and galvanic irritability of the nerves, and the faradic irritability of the muscle; while the galvanic irritability persists, is sometimes considerably increased, and is always changed qualitatively in a definite manner.

Before describing these changes in detail it will be well to briefly review the normal behavior of a nerve and muscle when excited by these currents. When an electrode attached to a faradic battery is

placed over a motor nerve and rapid interruption employed, if the current is of sufficient strength a tetanic contraction of all the muscles supplied by that nerve occurs. When slow interruption is used, clonic contractions occur synchronously with the opening and closing of the circuit, the contraction produced at opening being usually greater than at closing. If the electrode is placed over an individual muscle similar phenomena occur in that muscle. If, however, the current is strong or the muscles are small and close together as in the hand, neighboring muscles may also contract.

The *galvanic current* when allowed to flow through a nerve or muscle without interruption does not cause muscular contraction. If the circuit is *suddenly* opened or closed, and the current is of sufficient strength, contraction occurs at each opening and closure. If a nerve is being stimulated, all of the muscles supplied by it will contract, but if the electrode is placed over an individual muscle, this alone will respond unless the current is very strong, when neighboring muscles may also contract.

In using the *faradic current*, poles are not considered, but in the case of the galvanic, the pole used as the exciting or active electrode is of importance. The negative pole or kathode is the more stimulating, and therefore the weakest current that will cause contraction is when the circuit is closed with that pole over the nerve or muscle. If the current strength is increased, eventually a contraction will occur when the positive pole or anode is so used. For practical purposes opening contractions need not be considered, as the current strength required to produce them is too great. The kathode may be designated by the symbol *K* and the anode by *An*, Closure by *CL* and Contraction by *C*. This phenomenon may then be indicated as follows: *K CLC An CLC*. The contractions so caused are *short* and *quick*.

To *summarize*, it may be said that stimulation of a normal motor nerve or a muscle by either the faradic or galvanic currents causes a sharp, quick muscular contraction; in the case of the latter when the circuit is closed with the kathode as the active pole, the response is greater than when the anode is so employed. The behavior of nerve and muscle when a destructive lesion causing motor paralysis is in the central neuron is practically the same as above described, the exception being that where there is extreme hypertonicity with resulting spasticity of the muscles they may not be able to contract well for mechanical reasons and then there may be a pseudo-lessening of irritability.

When, however, a *destructive lesion* occurs anywhere in the peripheral motor neuron, as has been stated, a different behavior is observed. After a motor nerve is injured the following may occur: For the first day or two, the nerve, if not completely divided, may show some in-

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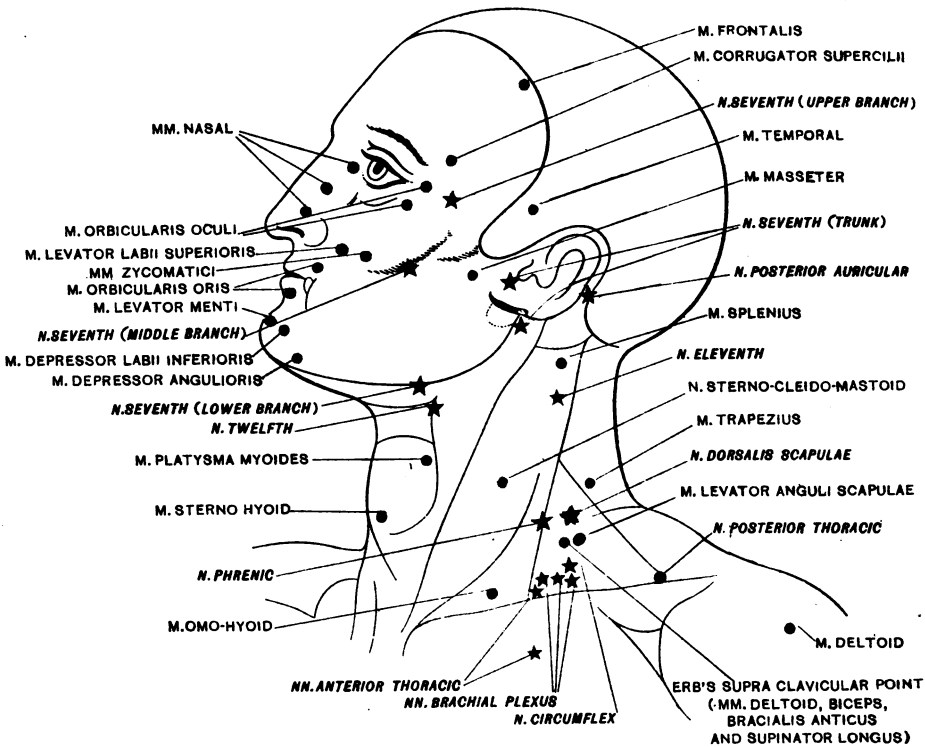

Fig. 149.

Motor Points of the Face and Neck.

(From Mosher's Electro-Diagnosis.)

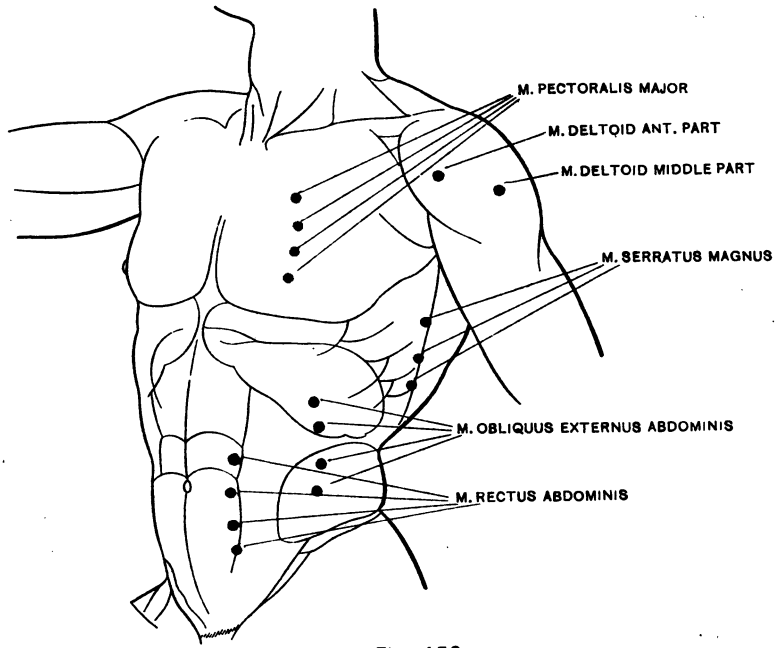

Fig. 150

Motor Points of the Trunk.

(From Mosher's Electro-Diagnosis.)

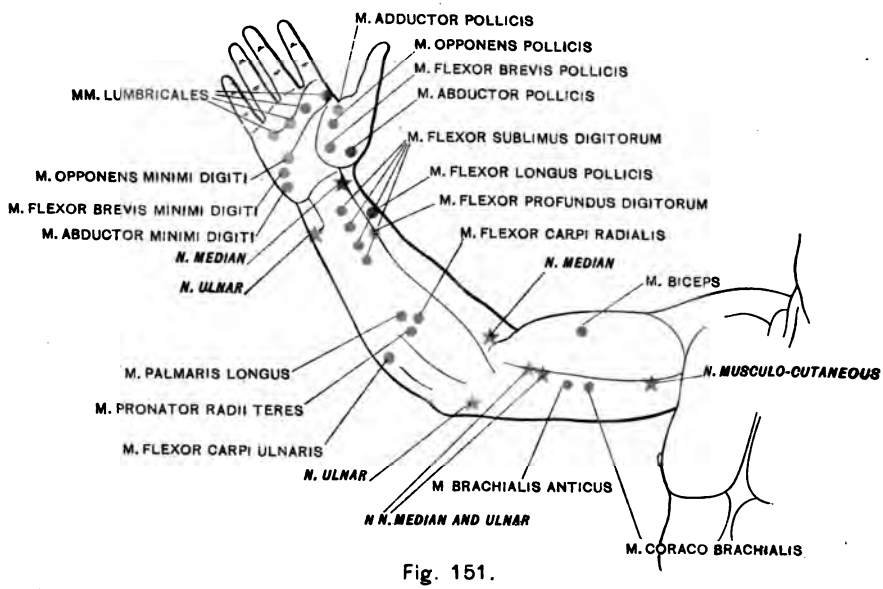

Fig. 151.

Motor Points of the Arm and Hand, Flexor Surface.

(From Mosher's Electro-Diagnosis.)

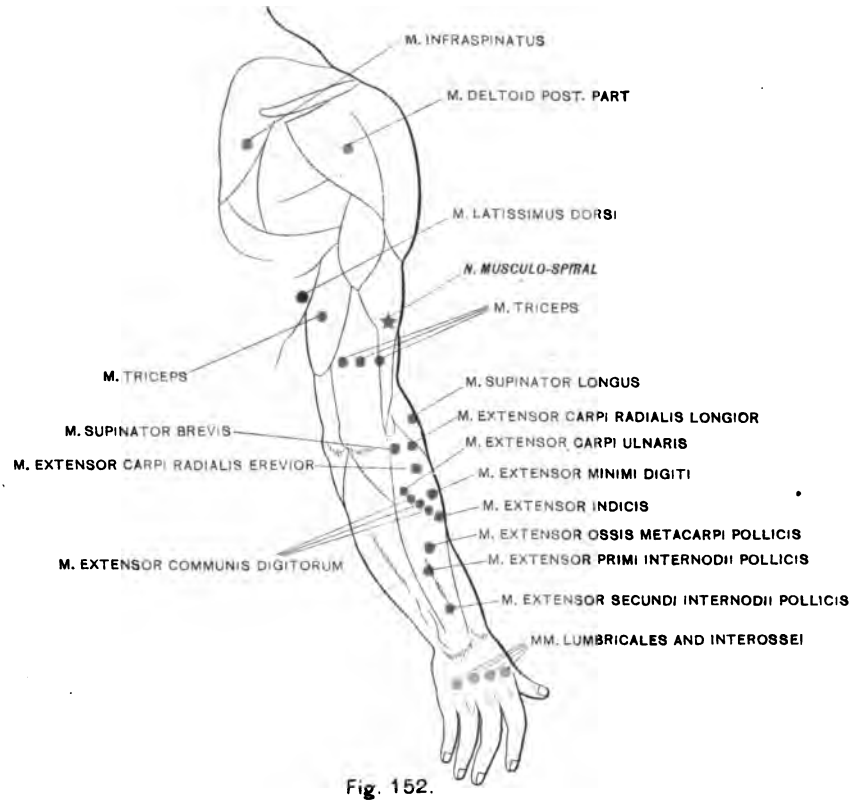

Fig. 152.

Motor Points of the Arm and Hand, Extensor Surface.

(From Mosher's Electro-Diagnosis.)

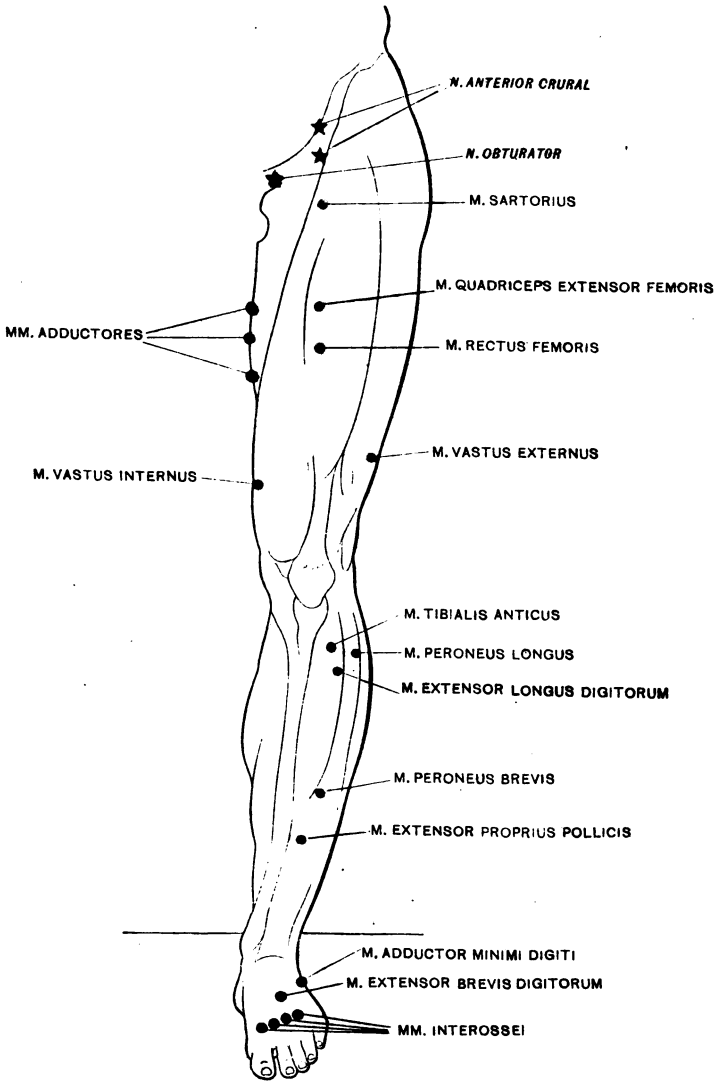

Fig. 153.

Motor Points of the Thigh and Leg, Anterior Surface.

(From Mosher's Electro-Diagnosis.)

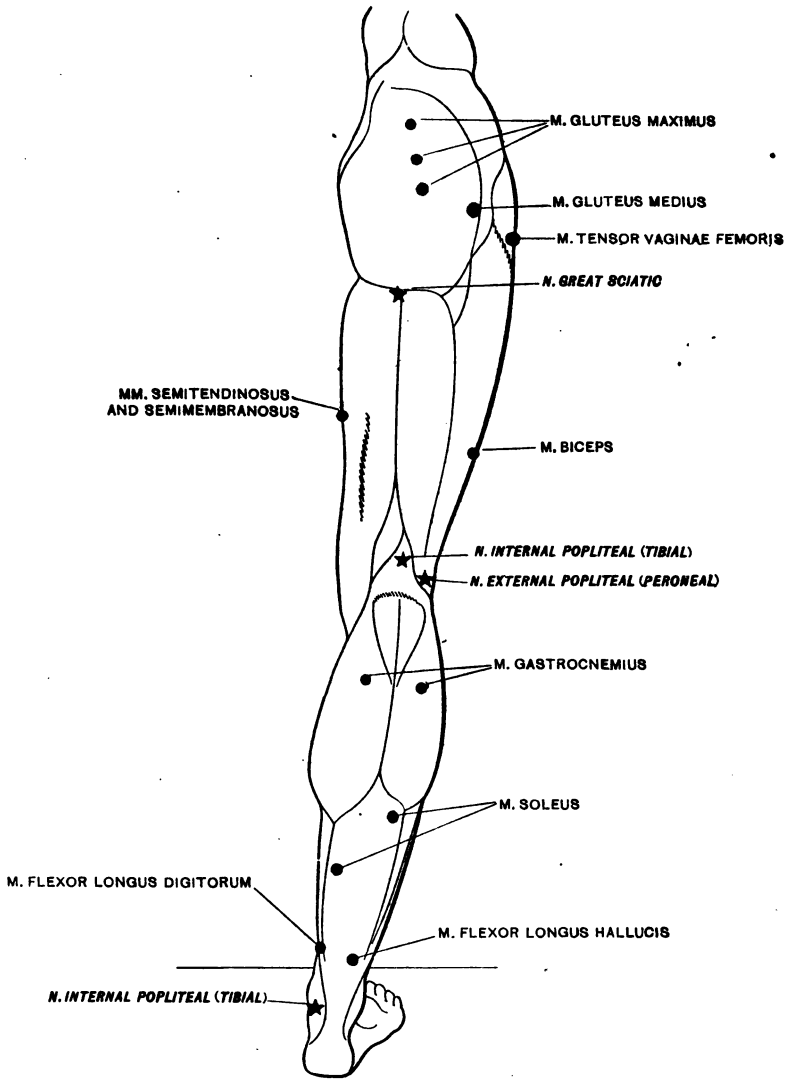

Fig. 154.

Motor Points of the Thigh and Leg, Posterior Surface.

(From Mosher's Electro-Diagnosis.)

crease in excitability when stimulated with either the faradic or galvanic currents, which is followed in a few days by diminution, and within a week complete loss. Usually, however, diminution begins at once. After 10 days or two weeks it will be noticed that the muscles when stimulated with the faradic current show diminution and eventually loss of irritability. If, however, the galvanic current is used a marked *increase* will be noticed, and the muscle instead of contracting with a short, quick jerk does so slowly and is more or less tetanic in character. This is known as the modal change. At the same time the *An CLC* increases and finally may become either equal to or greater than *K CLC*, so that the formula will read instead of *K CLC An CLC* (the normal formula) *An CLC K CLC* or *An CLC K CLC*. After a lapse of two or three months, the increased irritability diminishes and becomes less than normal.

The above is the fully developed DeR. It may not always be found, and the following modifications occur. The faradic excitability of the muscle may never be completely lost. In many cases *K CLC* remains greater than *An CLC*, although in such cases the latter is usually relatively greater than normal. These modifications are known as incomplete or partial DeR. The constant features are diminution or loss of faradic contractility and the slow contraction of the muscle excited by the galvanic current. Sherren has called attention to what he terms "the reaction of incomplete division." In this the muscles do not react to the faradic current, there is a slight increase in the galvanic irritability, the contraction is slower than normal, but not so much so as is seen in the typical DeR and *K CLC* is usually greater than *An CLC*.

In *testing for the presence of these changes*, the current from an electric-light circuit, which if alternating must be converted into the direct by a converter, is preferable; if not at hand dry cells (30–50), may be used. A means of gradually regulating the strength of the galvanic current is essential,[1] and if possible a milliampere meter should be in the circuit. This, however, is not essential. The indifferent electrode—by this meaning the one not placed over the nerve or muscle—should be large (3 by 3), if possible. The active electrode should be small.[2] The most irritable part of a normal muscle is where the nerve filament enters it. This is known as the motor point. They are shown in figures 149 to 154. In the degenerated muscle this is not the case and it is usually at the distal end or where the muscle fibers merge into the tendon. This has been termed "the longitudinal reaction." In small muscles this can not be very well determined, then the most irritable point must be searched for by moving the electrode about.

[1] The graphite type is best as it does not cut down the voltage.
[2] Larger ones can be used if the small size is not obtainable.

The patient should lie or be seated in a good light with the muscles perfectly relaxed. If one limb is being compared with the other, they should both be in the same position. It is well to use the faradic current first, as a normal contraction obtained by it at once shows that DeR is not present. It is of great importance in comparing the amount of contraction of the muscles of one side with the corresponding muscles of the other, that the current flows the same distance from the indifferent to the active electrode. The reason for this is that the further the current passes through the body the more resistance it encounters, and the greater the resistance the less will be the current strength of the active electrode. Therefore, if it does not flow about the same distance a greater contraction might be obtained on the side where the least resistance was encountered, while as a matter of fact the muscular contractility might actually be the same or less than that of the other side. A good plan is to place the indifferent electrode over the sternum. If one side only is diseased, we place the active electrode over the motor point of the corresponding healthy muscle and beginning at zero gradually increase the current strength until a contraction is obtained. The electrode is then placed in the motor point of the paralyzed muscle, and if a contraction is not obtained it should be moved to the distal extremity of the muscle and the current should be further increased until one is obtained or none can be obtained with a strength that the patient can bear. This will indicate either a lessened or absent faradic contractility and is sufficient in itself to show that a more or less developed DeR is present. The slowly interrupted current is preferable for this purpose although the rapidly interrupted current may be used; but if so, it must be interrupted by an interrupter on the handle of the electrode, or by alternately removing it from the body and placing it back again. Another possible source of error is due to the fact that after the current has passed awhile the skin under the electrode diminishes its resistance owing to the hyperemia produced, and hence the amount of contraction will increase. It must be remembered that if the current is very strong neighboring muscles that are healthy may contract, and this must not be mistaken for a contraction of the muscle being tested. If the fingers are placed lightly over it, contraction can sometimes be felt when it can not be seen.

Another method is to determine and record the amount of coil necessary to cause contraction. If one side requires more than the other there is lessened contractility on that side.

If it is not convenient to place the indifferent electrode over the sternum, an arrangement may be used first on the normal limb, then on the other. When the constant current is used the electrodes

must not be too close together or a pure polar action may not be obtained.

If a lessened or lost faradic contractility is noted, the *constant* or *galvanic current* should then, if possible, be used. A similar method to that above described is used, the amount of current needed to cause cathodal closure contraction of the normal muscle being first noted. This should be recorded by the number of milliamperes if a meter is in the circuit or by the number of cells required if there is not. The current should then be increased until an anodal contraction is obtained, which should be similarly recorded. The suspected muscle is then tested. It must be then noted if the contraction is slower than that of the normal muscle, if it occurs with a less strength of current and whether contraction occurs first with the anode; if not, if it is equal to the cathodal contraction, or if not, if it is relatively greater than that of the normal muscle as indicated by the strength of current necessary to cause contraction. These observations may be recorded on a chart similar to that devised by Mosher:

ELECTRO-DIAGNOSIS CHART.

Name_____ Date_____.
Electrical diagnosis _____
Treatment _____

Nerve or muscle.	Right.						Left.					
	Faradic.	Galvanic.					Faradic.	Galvanic.				
		CCc.	ACc.	AOc.	COc.	Character of contractio .		CCc.	ACc.	AOc.	COc.	Character of contraction.

As in using the faradic current, so with the galvanic, neighboring normal muscles may also contract, if strong currents are needed. In such cases it will be noted that the degenerated muscle contracts more slowly and that possibly its greatest contraction occurs with the anode, while with the other muscles it occurs with the kathode. If it is desired to test the nerve supplying the affected muscles, the active electrode should be placed over the nerve and the current moderately

increased. While a DeR or any of its modifications indicates a lesion somewhere in the peripheral motor neuron, such lesions may occur without its being present. Thus in progressive spinal muscular atrophy there is usually for some time a certain number of normal fibers in the atrophied muscle; if these predominate, their normal contraction may mask the abnormal. In muscles such as the deltoid, in such cases, one group of fibers may show DeR while another group reacts normally. In pressure palsies—those in which the nerve has been pressed upon from without, as sleeping with the head upon the arm—there is rarely DeR, although the faradic contractility may be somewhat lessened. The same may be said of cases of mild or subacute neuritis, especially when the inflammation is limited to the nerve sheath.

Bordier[1] has noticed that prolonged exposure to cold may cause DeR in the small muscles of the hand and feet, when no injury of the nerve is present. When the parts become warm the reaction becomes normal.

In the muscles of those suffering from *tetany* a greatly increased response may be obtained. This is equally so with both currents. There is no reversal of the formula. The muscles in *myotonia congenita* or *Thomsen's* disease show also an increased irritability to both currents and *An CLC* may be equal to or greater than *K CLC*. The presence of the faradic response at once eliminates the possibility of DeR being present. In *myasthenia gravis* there is an apparent diminution of contractility. This, however, is peculiar in the fact that at first the contraction is normal, but as it is continued it gradually diminishes until it disappears, owing to temporary exhaustion of the muscle. The *atrophied muscles* that are found about inflamed joints (arthritic atrophy), respond either normally or with a slight decrease. This is also true of the muscles in the various types of dystrophy or myopathy.

The determining of the electrical reactions in cases of *peripheral nerve* injury is of great importance. The diagnosis of such injury can be made usually without so doing, but its extent often can not be. If the reactions after ten days or two weeks are either of the partial type or that described as the "reaction of incomplete division," we can be sure that the nerve has not been completely destroyed or divided. On the other hand, the presence of a complete reaction does not always indicate that such is the case, as it may occur when the nerve is pressed upon by callus or inflammatory exudate. If for any reason it is determined not to expose the nerve but to treat the case otherwise, the absence of any return of function with the presence of a complete reaction at the end of three months would be an indi-

[1] *Bull., de l'Acad. de Med.* 1917, Feb. 6, p. 172.

cation to expose the nerve. The determination of the electrical reactions may also at times be of service in localized lesions in the spinal cord, such as hemorrhage or tumor. This depends upon the fact that lesions involving the gray matter of the cord or anterior nerve roots will cause the symptoms of a peripheral neuron lesion in the muscles supplied by the nerves that arise from the involved segments or roots.

In the case of peripheral nerve lesions the prognosis also may be guided by the character of the electrical reactions found. Thus, a favorable outcome may be expected when the reaction is incomplete, the time required varying from six weeks to three or more months as the reaction approaches the complete one in type. The presence of a complete reaction does not necessarily indicate that the patient can not recover, but it does mean that prolonged treatment will be necessary, whether this should be surgical or not depends upon etiology and concomitant symptoms. The quickening or approach to normal of the slow contraction is a favorable sign. It must be remembered that functional improvement sometimes may occur before there is much change in the character of the electrical response.

Vasomotor and trophic changes.—

I. *Trophic.*

(*a*) Skin: The skin may be dry, scaly, and cracked. It may become then smooth and shiny. It may show bluish or reddish discoloration. Ulcers may form, usually after some injury.

(*b*) The nails may be grooved transversely or longitudinally, split, brittle, curved and deformed. Hair may be brittle. Hypertrichosis is common.

(*c*) The subcutaneous tissue may become thick and edematous, sometimes suggesting a myxedematous appearance.

(*d*) The muscles may undergo extensive fibrosis, with fibrous contractures.

(*e*) Joints and bones may also suffer changes.

II. *Subjective sensory* disturbances accompany these various vasomotor and trophic phenomena in the forms of spontaneous pain or pain on pressure.

There is great variation in the degree of these vasomotor and trophic phenomena. Where present they may vary from slight neuralgic pains with cyanosis, to the intense and almost unbearable "causalgias" to be described below. The cause of the severe disturbances is not clearly understood. According to Mme. Athanassio-Bénisty, they are largely the result of arterial lesions with impaired circulation, super-added to the nervous lesions (in a way similar to Volkmann's contracture). According to Sherren and to

Tinel, they are the result of nerve irritation or neuritis of varying grade. Some of the symptoms are probably the result of involvement of sympathetic fibers.

Complete nerve division.—In complete nerve division the vasomotor and trophic phenomena are always relatively slight. There is some thickening and desquamation of the skin a few days after the injury. Somewhat later, the skin becomes dry, bluish pink, and trophic; a transverse groove may form in the nail, marking the date of paralysis. There is no sweating. There may be moderate cyanosis and edema. There is no pain or tenderness.

Incomplete division.—In some cases of incomplete nerve division, the vasomotor and trophic disturbances may be even less than those occurring in complete division. In other cases, apparently as a result of some nerve irritation or neuritic process, marked changes occur. There is spontaneous pain, which may be very severe; pain on pressure over muscles and nerve trunk. The painful area is often extremely tender, and the hyperesthesia may map out the full distribution of the injured nerve. The skin may be dry and scaly, or thin and glossy. Profuse sweats may occur, limited to the affected area. There may be marked changes in the nails, and the finger tips become thin and tapering. Fibrosis may occur in the subcutaneous tissues, muscles, and tendons, with resulting contractures and deformities.

In its most aggravated form, this condition was described by Weir Mitchell, Morehouse, and Keen during the Civil War, and given by them the name "*causalgia.*" Coming on a few days after the injury, and gradually increasing in intensity, it is characterized by smarting, burning pains, often of violent intensity, increasing at times to paroxysms as a result of movement, pressure, or even emotion. The pain may be so extreme as to be almost unendurable. The skin is mottled, smooth, and glossy (Mitchell's "glossy skin"). Extreme hyperesthesia of the skin and hyperidrosis are common and may extend well beyond the cutaneous region of the affected nerve. The median and sciatic nerves are those most commonly affected.

III. *Objective examination.*—Objective examination of the nerve: (*a*) Pressure over the injured nerve trunk is generally painless. This is practically always the case in complete division. In certain cases of incomplete division with irritation, the nerve may be painful on pressure over the entire trunk below the level of the lesion. Along with tenderness of the nerve trunk, there is tenderness of the muscles supplied by that nerve.

(*b*) Formication or tingling on pressure. Compression or light percussion over an injured nerve trunk often produces a tingling sensation, likened by the patients to electricity, and referred to the cutaneous distribution of the nerve. Tinel lays great stress on this sign, and considers it the most important means of following the

progress of a given case. He believes that wherever it occurs it indicates the presence of young axis-cylinders in course of regeneration. Generally it appears from four to six weeks after the injury. This tingling is quite different from the pain produced by pressure over an irritated nerve. The latter is characterized by being greatest at the point of pressure and is associated with even greater pain on pressure over the muscles it supplies.

As to the significance of the sign. According to Tinel, if this tingling, produced by pressure over the nerve, and referred to its cutaneous distribution, remains fixed at a given point, say at the site of the lesion, and can be produced only from this point, and not from any point further down on the course of the nerve, it means that the regenerating axis-cylinders have met an impassable obstacle, and are matted together to form a bulbous neuroma. If, on the other hand, we find the sign present at the site of the lesion, then in a week or so we can elicit it from a point further down along the course of the nerve, then we have evidence that that nerve is regenerating and its fibrils growing toward the periphery. Tinel states that the course of progress of the nerve may be followed week by week, till the periphery is reached. As formication on pressure moves further and further along the nerve it disappears at the site of the lesion above.

The evidence of downgrowth of the nerve is of equal value, whether we are dealing with spontaneous regeneration, or regeneration following nerve suture.

Clinical syndromes.—We have described in a general way the various phenomena which follow nerve injuries. These changes may be grouped together in the form of more or less well-defined complexes, or syndromes. As was stated at the outset, it is impossible from our examinations or even sometimes at operation to determine the exact pathological condition, and so we are compelled to group our symptoms according to clinical rather than anatomical criteria.

A. *Symptoms of complete division*—I. *Motor*—(1) Complete paralysis of all muscles supplied by the nerve (below the site of the lesion).

(2) Progressive loss of muscle tone.

(3) Progressive atrophy (beginning two or three weeks after the injury).

(4) Loss of reflexes.

II. *Sensory.*—Complete loss of epicritic, protopathic and deep sensations over the region supplied by the nerve.

III. *Reaction of degeneration*—especially the slowing of the galvanic contraction.

IV. *Vasomotor symptoms*, moderate dryness, and scaling, atrophy of the skin, slight cyanosis, etc.

V. *Examination of the nerve.*—Absence of spontaneous pain, or pain on pressure over nerve trunk and muscles.

This group of symptoms, occurring together, means complete anatomical or physiological division of the nerve. This does not mean that spontaneous regeneration is impossible. In the complicated injuries produced by war wounds, however, with extensive scar tissue formation, spontaneous regeneration is unlikely. For the full development of this picture, two or three weeks may be required.

B. *Incomplete division without irritation.*—The clinical signs of complete division at a given level are fairly constant. The clinical signs of incomplete division of the same nerve at the same level vary greatly according to the nature and severity of the lesion in each case.

I. *Motor.*—(1) Weakness of some or all the muscles supplied. This may be a loss of function so slight as to readily escape notice; or it may be an almost complete paralysis; or it may be a weakness of some muscles, a paralysis of others; (2) diminution in muscle tone. Usually the hypotonia is much less than in complete division. (3) Atrophy. This develops more slowly and is less intense than in complete division. (4) Reflexes. Diminished or lost.

II. *Sensory.*—The sensory loss may be represented by: (1) A scarcely recognizable epicritic loss; (2) complete epicritic loss, with preservation of protopathic; (3) loss of epicritic, protopathic and deep.

III. *Electrical reactions.*—Here again we have a series of changes, from the slightest quantitative reduction, to almost complete division: (1) Reduction of faradic irritability of muscle; (2) loss of faradic irritability; (3) beginning slowing of galvanic contraction. As will be recalled, Sherren's reaction of incomplete division consists in loss of faradic irritability, increase in galvanic irritability, some slowing of the galvanic response, although the response is quick as compared with that of complete division.

IV. *Vasomotor and trophic.*—These are slight; in the mildest cases, absent.

V. *Examination of nerve.*—No spontaneous pain, or pain on pressure over nerve trunk or muscles.

According to J. R. White, if the lesion is uniformly distributed through the cross section of the nerve, the following series of changes indicates in general an increasing severity of the lesion: (*a*) Loss of epicritic sensibility, partial or complete; (*b*) loss of protopathic sensibility; (*c*) loss of motor power; (*d*) loss of faradic response.

If, as often happens, the cross section of the nerve is irregularly involved, other factors enter and completely change the order. The irregular type of lesion will be discussed (under dissociated symptom-complex) later.

C. *Incomplete lesions with irritation.*—Here we have the same phenomena as those just described (B), and the same variations in degree.

The vasomotor, trophic, and subjective sensory disturbances distinguish incomplete division with irritation from incomplete division without irritation. Within a few days after the injury, pain develops, gradually increasing for two or three weeks. There is hyperesthesia of the skin and tenderness on pressure over nerve trunks and muscles.

The skin may be dry and scaly, or smooth and shiny. There may be profuse sweats. Trophic changes are found in the nails. Hypertrichosis may occur. There may be fibrous infiltration of subcutaneous tissue, muscles, tendons, and joints, leading to contractures and permanent deformities.

In many of these cases spontaneous regeneration of the nerve occurs, with restoration of sensation and motion. But even after complete regeneration, function may remain seriously impaired as a result of the fibrous contractures and deformities.

The phenomena above described vary in degree. They may be slight, indicated perhaps only by a little pain and a little fibrosis of the muscles, or they may reach the unbearable intensity of a "causalgia."

D. *Dissociated syndromes.*—Injury to a nerve need not affect it uniformly throughout its cross section. Half the nerve may be divided, and the other half be normal, or half the nerve may be divided and the other half partially compressed. All possible combinations may occur, especially in larger nerve trunks, like the sciatic. As a result of partial injuries of this type it has been found that, as Stoffel maintained, peripheral nerves have an inherent funicular topography. That is, in the cross section of a given nerve, there is a more or less constant and permanent arrangement of fibers. A group of sensory fibers lies in one zone; another group of sensory fibers for another region in another zone; the fibers for a certain group of muscles in a third zone, etc. Many of the facts regarding the inherent funicular structure of the various nerves have already been investigated. Their study is important, for we may be able to determine clinically that, while one part of the cross section of a nerve is divided, another portion is intact, or merely slightly injured; and surgical procedures can be directed toward only the severely damaged portion, avoiding, for example, the need of dividing and resuturing the entire nerve.

E. *Ascending neuritis.*—Ascending neuritis is a rather uncommon but serious complication of nerve injury. Starting from an area of infection—usually a suppurating focus in the bone—it gradually works its way up the nerve trunk, into the plexus and even to the

spinal cord. It is characterized by severe spontaneous pains, referred to the whole distribution of the nerve—even to parts above the lesion. The nerve trunk is tender on pressure. As the process ascends, there is an ascent of pains, and of the tenderness over the nerve trunks. There is an ascending advance of muscle atrophy and objective sensory disturbances.

Ascending neuritis may heal spontaneously or after the clearing up of the infectious focus, or it may continue for months or years, causing serious permanent disability. It has been necessary at times to divide the nerve high up or to amputate the limb. But even these heroic measures do not always stop the process.

F. *Reflex contractures.*—Under this name Babinski and Froment have described a group of contractures, paradoxical in type, which

FIG. 155.—Contracture of the hand from slight lesion of the ulnar in the forearm. (Contracture of the interossei and of the hypothenar eminence.) (Tinel.)

bear a distinct resemblance to hysterical or functional contractures but which these authors believe should be separated from the hysterical group.

In all these cases there are some signs pointing to the existence of an organic lesion. Vasomotor and trophic disturbances, pain and sensory disturbances; muscular hypertonia, symptoms in the main suggesting nerve irritation.

But associated with these relatively slight signs of organic lesion are contractures out of all proportion to the possible extent of the lesion. As a rule, they are paradoxical in type, contractures exactly opposite to what one would expect as a result of a paralytic lesion of that nerve.

The contracted muscles offer a greater or less resistance to attempts at reduction. After having persisted sufficiently long, the deformities tend to become permanent by the overstretching of the antagonistic muscles.

Generally speaking, these contractures are limited to the region of the nerve involved.

So we have in these cases an injury which is always relatively slight, followed by definite but slight evidence of organic injury of the nerve, by contractures paradoxical in type, generally limited to the region of the affected nerve, but out of all proportion to the degree of damage.

FIG. 156.—Contracture of the hand owing to slight lesion of the ulnar in the forearm. The patient can bend only his thumb, index and middle fingers, and even these very imperfectly. Passive flexion is fairly easy. This figure represents the maximum of flexion possible. (Contracture of interossei and extensors.) (Tinel.)

It would seem that pictures of this type are due to an irritative condition of the nerve (a slight neuritis) with a large superimposed functional element.

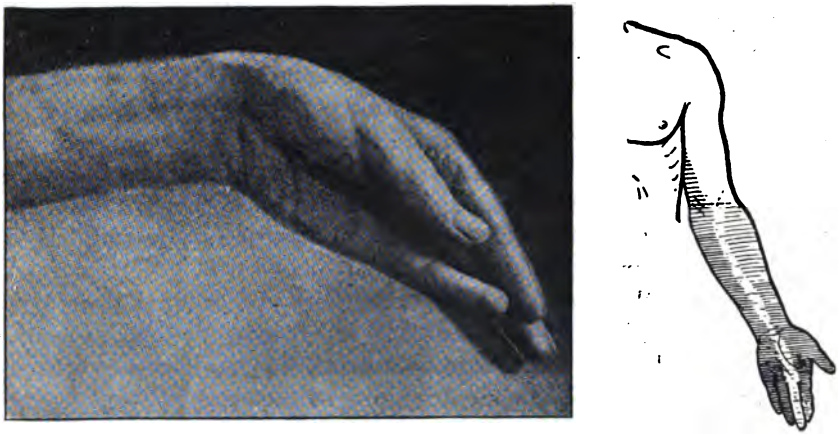

FIG. 157.—"Accoucheur's hand" due to contracture of the muscles of the hand; after a fall producing fracture of radius. Thermal and vaso-motor disturbances, profuse sweats. Hyperesthesia on the region of C6, C7, and D1 (probable wrench of the brachial plexus). (Tinel.)

Regeneration of nerves.—As was stated before, after division of a peripheral nerve, the distal segment degenerates. Before the function of that nerve can return, regeneration must take place from the site

of the lesion to the peripheral end-organ. This can occur only as a result of the down-growth of axis-cylinders from the central nerve stump. Cases have been reported where immediate return of function followed the suturing of a severed nerve; but such reports are undoubtedly based on faulty observation or misinterpretation. Once a nerve is so injured that its distal portion degenerates, function can only return after a considerable interval, depending on the distance which the nerve fibers have to travel to reach their end-organs. The first signs of regeneration rarely appear under four to six weeks.

The order in which the various functions return and the time required for each, are subject to such variations that exact data are impossible. Perhaps the first sign of functional recovery is an improvement in muscle tone. There is an increased firmness of the muscle on palpitation. Following this after a variable interval there is a return of muscle sensibility.

Cutaneous sensibility returns somewhat later. From the elaborate studies of Head and Sherren on the return of cutaneous sensibility, we learn that the return of protopathic sensibility begins in 6 weeks to 6 months, and is complete in 6 to 12 months. The epicritic return begins in about 6 months and requires a year or even much longer to become complete.

Return of motion begins in perhaps six to nine months, depending on the distance of the point of suture from the periphery. The muscles nearest the seat of injury are the first to regain voluntary power. This return of voluntary power comes about very gradually and may require two or even three years for its completion. As regeneration progresses, we find changes in the electrical reaction. Gradually the galvanic hypoexcitability improves; polar inversion becomes polar equality, and a qualitative change takes place on the galvanic contraction, so that it gradually loses its slow character and approaches the normal. The final stage in recovery of the electrical reactions, return of faradic irritability, is rarely complete till voluntary movement has returned.

Along with the functional recovery, there is a gradual clearing up of various trophic disturbances. It is always possible, of course, that certain deformities due to overstretching, or fibrosis with contracture may remain permanent in spite of the complete regeneration of the nerve.

Finally, there is the symptom of formication (i. e., tingling produced in the cutaneous distribution of a nerve, by pressure over the nerve trunk). This has been described under the methods of examination. Tinel considers it the most important of all the signs of nerve regeneration. Beginning four to six weeks after the injury,

its presence, according to this author, means the presence of growing axis-cylinders. As the new nerve fibrils grow down, their course may be followed by the progressive advance of the zone within which formication can be produced. In this way it is possible to follow the progressive advance of the axis-cylinders—to know whether they are advancing, and how rapidly. If the zone over which the sign of formication can be obtained becomes stationary and remains in one place for a considerable time (several weeks) it means that the progress of regeneration has closed.

The above discussion of regeneration refers particularly to cases of complete anatomical or physiological division. If the lesion is incomplete, the course of regeneration will vary greatly, depending on the extent of the damage and its distribution over the cross section of the nerve. Recovery of function may begin within a few days. In contradistinction to the findings in complete division, epicritic and protopathic sensibility may recover simultaneously.

Root and peripheral nerve lesions.—The question may arise, especially in the case of lesions of the brachial plexus, or of the lumbar plexus, whether we are dealing with a lesion of a nerve root, or a lesion of a peripheral nerve. The differential diagnosis between the two must depend on the grouping of the muscle involvement, or the distribution of the sensory disturbance, or both. To give an example: In the lower-arm type of lesion of the brachial plexus we have a lesion of C8 and D1, or the primary cord resulting from the union of these roots. A lesion here will cause paralysis and atrophy of all the small muscles of the hand, and certain flexors of the wrist. If we compare this with a lesion of the ulnar nerve, which arises from this cord, we find a difference. There is the same weakness of the inner flexors of the wrist. Many of the small hand muscles are paralyzed and atrophic; but some of the small muscles of the thenar eminence receive their nerve supply from the median, and these muscles are spared.

In the root lesion, the area of anesthesia would extend along the inner aspect of the arm and hand, whereas in the lesion of the ulnar it would confine itself to the hand.

Spinal cord and nerve roots.—At times it may be very difficult to distinguish between damage to the spinal cord, and damage to the nerve roots. If a spinal cord lesion is strictly confined to the anterior horns, we have paralysis, without sensory disturbance. It will be impossible to distinguish this from a lesion of the anterior root. It will be readily distinguished from a lesion of the root after the anterior and posterior portions of that root have joined. On the other hand, if a spinal cord lesion is sufficiently extensive to produce both a motor and a sensory disturbance at a given level, it will almost

certainly produce distant symptoms, by interruption of the long descending and ascending spinal tracts. There will be spastic paralysis, and a dissociated sensory disturbance below the level of the lesion.

Central and peripheral lesions.—In paralysis due to brain lesions, the signs will differ completely from those due to injuries of peripheral nerves. The disturbance is of the upper motor neuron type, and is characterized by (*a*) hypertonicity and spasticity; (*b*) increased reflexes, perhaps with ankle clonus, patellar clonus, and Babinski; (*c*) absence of muscle atrophy; (*d*) normal electrical response.

Hysterical or functional disturbances.—It is frequently necessary to distinguish between organic lesions of peripheral nerves and functional disturbances. Whereas this differentiation is simple in a clear-cut case of either type, there are many cases of organic injury with superimposed functional elements, in which it is exceedingly difficult to say just how much is organic and how much functional. We can give the conventional points of differentiation, but realize that they do not answer all requirements.

In a typical hysterical case the paralysis is often out of all proportion to the degree of the lesion; it is extensive and absolute; it involves a limb or segment of a limb, without any respect to the anatomical relations between a given nerve and the muscles supplied by that nerve. The paralyzed muscles show no atrophy; there is no change in the reflexes; there is no disturbance in the electrical reactions. Anesthesias are often present. Like the paralyses, they are no respectors of anatomical laws. They are often extensive, complete, and involve all modalities of sensation. They are apt to take on the glove or boot type, with a sharply demarcated upper boundary, or, if they reach the mid-line, stop exactly at the mid-line. The limb is useless, and the patient makes no effort either to use it or to compensate for the paralyzed movements by substitution movements. When told to move the limb, the patient either makes no effort, or else contracts both the "paralyzed" muscles and their antagonists, so that no movement results.

After a functional paralysis has existed for a long time, there may be slight changes in the part due simply to disuse, slight cyanosis, slight diminution of volume, and even slight reduction in electrical irritability.

In addition to these gross disturbances other types of functional paralysis occur. After prolonged immobilization following injuries of bones or joints, the patient may show an apparent paralysis of the muscles, simply as the result of the habitual disuse, akinesia amnestica. No other signs of nerve involvement are found, and there is prompt restoration of function with appropriate treatment. A

similar condition may result from pain, the thought of the pain on movement being sufficient to inhibit movement.

Difficulty in diagnosis is especially apt to occur in nerve lesions of the irritative type. These may produce contractures and positions more or less paradoxical. Certainly in the "reflex contractures" of Babinski and Froment it is hard to say just what per cent of the disturbance is functional.

After-results of nerve lesions.—It is necessary to differentiate between paralysis due to a nerve lesion and an apparent paralysis which may be the aftermath of that lesion. A nerve which has been injured may completely regenerate, and yet the return of function may be lacking. This may occur when the paralyzed muscles have been so overstretched by faulty position that muscle contraction merely succeeds in taking up the slack and no movement results. Or the position of the part may be so fixed by fibrosis and contracture of the antagonists that the resistance can not be overcome; or, in long-standing irritative lesions, the paralyzed muscles themselves may have undergone extensive fibrosis or their tendons may be bound by adhesions.

In all these cases, in spite of complete restoration of nerve conductivity, function is impossible. Such a case must be treated by orthopedic measures, not by measures directed toward the nerve.

Lesions of bones, joints, and muscles.—Loss of muscle function may be due not to a lesion of the nerve but to purely mechanical causes, such as fractures, dislocations, ankylosis, inflammatory conditions (e. g., myositis, abscess), or to actual destruction of the muscle substance by the injury.

Ischemic paralysis (Volkmann's paralysis).—Ischemic paralysis generally results from obliteration (thrombosis, ligature) of one of the larger arteries of the extremities, or from the application of a bandage applied too tightly and left in place too long.

The mechanism of its production is not clear. We do not know why, in one case, ligation of the artery will produce only slight circulatory disturbances, in another case an extensive ischemic paralysis. The condition of the artery, the collateral circulation, disturbance of the sympathetics, and concomitant lesions of peripheral nerves, all may play a rôle, but do not explain the development of the condition in an individual case.

There are two stages in its development. At first there is edema, cyanosis, and cooling of the part. The patients complain of pains and paresthesias, which may be very severe.

After a few weeks the edema diminishes, and there is a progressive fibrous transformation of the tissues which eventually results in conversion of the part—subcutaneous tissue, muscle, and tendon—

into a fibrous mass. There is a progressive loss of motion, due to fibrosis of the muscles, but some degree of motion may persist for a long time. There is a variable degree of anesthesia, greatest at the periphery and gradually diminishing proximally. Electric examination shows an intense reduction in excitability; but there is rarely a true R. D., and such reaction as is obtainable can generally be elicited by stimulation of the nerve trunk, as well as by direct stimulation of the muscle. The pulse is often diminished.

Ischemic paralysis may develop to a varying degree; at times it may be difficult to differentiate from a nerve lesion, especially an irritative lesion with contracture. The important differential points are reduction of the pulse, absence of R. D. in spite of extensive atrophy, and the sensory disturbance which fails to correspond to nerve distribution.

Treatment.—Whenever a nerve is injured, all the structures supplied by it begin to undergo changes; and the effects of these changes must be prevented as far as possible.

The paralyzed muscles tend to stretch; and the graver the paralysis and the longer it lasts, the greater the stretching. When this stretching has become pronounced, even complete regeneration of the nerve will not suffice for the return of function. The muscle will contract, but in contracting will merely succeed in taking up the slack in its tendon, and not in causing movement. Coincident with the lengthening of the paralyzed muscles, there is a gradual shortening of their antagonists, due to a constant unopposed action. If this lasts long enough, the antagonists will undergo fibrous changes and the shortening will become permanent.

Both these evils can be prevented in the same way, and this is one of the first essentials in the treatment of nerve injuries, namely, by placing the paralyzed limb in such a position that deformity can not occur. Thus in a musculo-spiral palsy, the wrist must be put up in hyperextension, to prevent elongation of the extensors and shortening of the flexors. In peroneal palsy, the foot must be put up in exaggerated dorsal flexion.

A muscle which is paralyzed is inactive. The circulation is slow. Trophic changes occur, and if it remains inactive long enough, there is a gradual fibrosis. When such fibrosis is far advanced, complete regeneration of the nerve will not result in restoration of function. The regenerated nerve fibers find nothing but scar tissue on which to act. Changes of this type can be largely prevented by supporting the circulation and by keeping up muscular activity. This may be accomplished by massage and by electrical treatments. Every paralyzed muscle should have daily massage. Every paralyzed muscle should be made to contract daily by means of the electric

current. It is necessary to produce only a few weak contractions. Too active or too prolonged treatment may do more harm than good.

As to the form of electricity used, it should be that form capable of obtaining the contractions with the minimum of discomfort. If the muscles react to the faradic current, faradism should be used. If not, galvanism. It will often be found that a degenerated muscle will respond very poorly to the galvanic current when applied over the motor point, but will respond much better if a point of stimulation is chosen well down toward the tendinous insertion of the muscle (so-called displacement of the motor point, or longitudinal reaction).

Even better than the galvanic current in many cases is the use of condenser shocks rhythmically interrupted by means of a metronome. This is often less painful and in their highest powers (2.5 and 3.0 microfarads) cause contraction of muscles which fail to respond to galvanism.

In addition to changes in the paralyzed muscles and their antagonists, we may have changes in joints and tendon sheaths. Persistent immobilization readily leads to adhesions. These will not be cured by subsequent regeneration of the nerve. They may be prevented by frequent mobilization of the part, passive motion, and massage.

The essential of treatment is prophylaxis. Muscles, joints, and tendons must be kept mobilized and in good condition until regeneration of the nerve can occur.

The irritative lesions, those with pains, and a tendency to hypertonicity and early contracture of the affected muscles, require sedative treatment of various kinds. Among these measures are prolonged warm baths, galvanic baths. The positive pole of the galvanic current has definite sedative qualities, and prolonged baths of this type with weak currents (3–4 milliamperes) are very soothing. Similarly the faradic current, or combinations of the two, may be used. Radiotherapy is also recommended. These treatments may be followed by gentle massage and passive motion. In the more serious irritative cases, as the causalgias, it may be impossible to obtain relief by any of these measures, or even to carry out such measures as massage, because of the pain.

In all cases of nerve injury, and especially those with extensive anesthesias and trophic disturbances, slight traumata are apt to produce serious results. The parts must therefore be protected. Care must be exercised to avoid burns from too hot baths or too strong electric currents.

Finally, voluntary movements and prescribed daily exercise for the injured muscles, as power returns, are an important adjunct to treatment, both for keeping these muscles in condition, and for the

mental effect on the patient. Without them, prolonged immobilization is much more apt to result in the development of functional disturbances, superimposed on the existing organic condition.

Indications for operation.—Immediate suture in war wounds is rarely possible, because nearly all are infected. In war surgery many additional problems arise, due both to infection and the extensive destruction of the tissues. The loss of nerve substance is frequently considerable and the amount of scar tissue present excessive.

It is a fundamental principle of nerve surgery that all operations must be done in a sterile field and in the absence of the possibility of infection. Hence secondary suture is the rule. Recrudescence of infection in an apparently healed wound must always be considered. More especially is this true when there has been extreme comminution of a fracture, or when there remain in the field scattered fragments of metal. The presence of these in an X-ray picture should mean a longer delay than might otherwise have been deemed expedient. These fragments are many times found at operation walled off and surrounded by muco-purulent fluid. When broken into, such foci of infection may light up and the aim of the operation be annulled by increasing scar formation, both by another infection and by the insult of an additional operation. Therefore, sufficient time must elapse to insure a clean, non-infected field.

The aim of surgical interference is to facilitate the normal process of repair by removing all causes hindering the down-growth of the neuraxes and to facilitate their growth either through normal paths, i. e., by end-to-end suture, or down newly furnished paths, be they nerve transplants or artificial conducting paths.

Since secondary operations only are permissible, at what period should they be undertaken? The first consideration is, as we have said, a sterile field. The second consideration is the nature of the trauma and the progress of the nerve injury. Not all nerve wounds require operative intervention. A large percentage recover without operation, variously placed at from 40 to 60 per cent. However, it is but just to state that in a considerable proportion of these the time of convalescence might have been materially shortened by a comparatively slight operation.

Unfortunately, with the exception of partial and incomplete nerve injuries, it is impossible to determine, except by waiting a sufficient length of time for evidences of nerve regeneration to manifest themselves, whether a nerve will recover without operation. Therefore, it is of the greatest importance to study at definite intervals all available data and to be governed by the changing picture thus collected. The progressive changes are more important than the findings at any one particular time. For this reason it is essential to

have a report of the successive findings in any given case. The signal importance of early and frequent examination can not be overemphasized; for it is only by such measures that one can become cognizant of the changing pathology of the lesion. Only by careful, consecutive examinations can proper and adequate treatment be outlined and needless delay prevented.

It is an axiom of peripheral nerve surgery that so long as there are progressive signs of nerve regeneration no surgical interference is permissible. What, then, are the evidences of nerve regeneration? The earliest and most reliable are: Formication, shrinkage of areas of anesthesia, return of deep sensibility, and return of muscle tone and muscle sensibility. Of these, perhaps the most important and interesting is formication. This sign is elicited by gentle pressure upon the nerve trunk, producing thereby a peculiar creeping sensation referred to the peripheral distribution of the nerve. The progressive regions along the nerve at which this phenomenon can be elicited is an excellent guide for the progressive down-growth of the neuraxes. Shrinkage of the areas of anesthesia is a reliable early index of regeneration. The concentric contraction, from above downward, of the borders of loss to pin prick and temperature sense, specially extreme degrees of temperature, are early signs of nerve repair. Frequently there occurs dissociation of temperature sense; extreme degrees of both hot and cold being confused, hot being appreciated as such one time and recognized as cold the next instant, and vice versa. Deep sensibility often returns earlier than other forms of sensation. The return of muscle tone and muscle sensibility occur at about the same time. There are other signs of nerve regeneration which manifest themselves further in the progress of recovery. The above signs of regenerating nerves give a very good index of the progress of their repair. As long as there are signs of progressive regeneration, operative intervention should not be undertaken. When these have ceased to advance, or when, after due and repeated examinations, they have not appeared, surgical interference is definitely indicated. In from three to four months some evidences of nerve regeneration should manifest themselves. By waiting this long, little time is lost, since during the greater part of the intervening period the wound has been infected.

Surgical procedures.—The time of operation will then depend on the progress of the case and the probability of a sterile field.

Considered from a surgical standpoint alone, the earlier the operation is undertaken the less difficult it is, the clearer the anatomical field, the better the operative end-result, and the more complete the recovery. The longer the delay, the greater the amount and density of the scar tissue and callus; the more bound and

fixed, in a retracted position, becomes the nerve trunk; and the denser the connective tissue within the nerve. Furthermore, the longer the delay, the greater the secondary changes in the muscles and tendons, etc. Hence, from a purely surgical standpoint there is every justification for early interference. The well established fact that a certain percentage of nerve lesions recovers without operation offers the only valid objection to early surgical intervention. However, which cases will recover and which will not recover can be determined only by awaiting evidences of regeneration. Rather than remain inactive too long, an exploratory incision should be made in certain cases. Unfortunately, by the most careful examination it is now and then impossible to distinguish before operation those nerve lesions which present anatomical loss of nerve substance from those with interruption of conductivity without loss of anatomical continuity. When complete, both manifest themselves alike. By early exploratory operation those cases would be recognized which could not offer any possible hope of recovery without surgical interference. Therefore, in selected cases careful exploration is indicated.

No surgical procedure should be undertaken without electrical study, both before and during operation. Nerve operations are operations of choice and should be done where there are both ample facilities and ample time for thorough study.

A general anesthetic should always be used. When the patient is anesthetized and before the incision is made, the extremity involved should be freely mobilized and adhesions broken up, so that the part may be placed and held in proper position after operative treatment. If there are contractures these should be corrected in so far as is possible before operation. Correct pre- and post-operative treatment, unless properly carried out, would very likely annul the success of the operation.

An absolutely dry field is one of the most important conditions of surgical technic. The use of the tourniquet is to be avoided, because each bleeding point should be dealt with individually. Kocher's blood platelette preparation, if available, is very helpful in checking capillary oozing within the scar tissue. A weak solution of adrenalin also gives excellent results. Hemorrhage from the nerve ends is often profuse and is best controlled by gentle and prolonged digital pressure. Bits of torn muscle held over the nerve ends will be found advantageous in checking hemorrhage. Blood clots between the nerve ends increases materially the amount of connective tissue formation.

In wounds with the nerve imbedded in much scar and callus it will be found safer to dissect from above down and below up; from sound and normal tissue into scar. Much time will be wasted and

damage done by searching in the scar for the divided nerve ends. The distal end often is small, frayed, and diffused in the surrounding scar. Care must be taken in freeing the nerve not to cut nerve branches.

When the nerve is free from the immediate scar it can be freed and liberated, in its contiguous parts, by gentle traction. This is best done before excising the intervening scar. This admits of using the scar to make traction, thus avoiding additional trauma to the nerve trunk or nerve ends. Squeezing the nerve ends to make traction must be avoided.

It is good procedure to cut only partially through the scar, using it as a means of fixation for the nerve ends. Two things are thus gained—firstly, it is easier to suture when the ends are fixed; secondly, by so suturing there is less tendency to axial rotation, and consequently greater accuracy in maintaining the internal topography of the nerve. The more accurate the union, the more complete and the earlier the return of function. However, it is many times impossible to attain union of divided ends with due regard for the internal anatomy of the nerve. Accurate juxtaposition should be nevertheless the aim toward which the operator should strive.

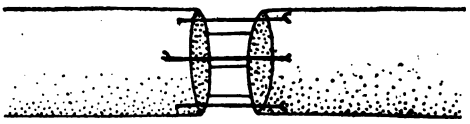

FIG. 158.—Illustrating nerve suture. Catgut is employed on fine non-cutting needles. Three to four sutures placed through the epineurium will usually suffice. The cut ends should be approximated, but not forcefully. The nerve stumps should not be rotated when the sutures are tied Non-axial rotation is essential to successful suture. Cf Stoffel's figures. (Lewis.)

Small, smooth, round, half curved needles with either fine silk or plain catgut should be used. For smaller nerves, such as transplants, the finest silk is to be preferred. It is of advantage to wax the silk sutures before they are placed. The suture should include as little of the nerve as possible. The nerve ends should be brought gently into alignment without pressure or tension. Care should be taken not to handle the nerve ends with forceps. A small amount of connective tissue will always form between the nerve ends. The thinner the layers of connective tissue, the less resistance will the neuraxes meet and the more complete the regeneration. A method of nerve suture which is to be especially commended is as follows: Three sutures are equidistantly placed, each including a little more than the epineurium (this is better than to include too little tissue, for then the stitches are very apt to tear out and still greater damage be done in an effort to resuture). The suture ends are clamped and not tied until all three are in place. Each one acts as a guy suture in the placing of the next suture. If necessary, a primary stay may be used until the other three are in place and tied. This stay suture may also be of assistance in preventing axial rotation.

The type of operation depends on the anatomical findings, considered together with the clinical picture and the results of successive examinations. It is often impossible even at operation to determine whether or not nerve axes have reached the distal stump. If the axes are new and have not, as yet, reformed the motor end plates, then, even though they have reached the distal stump, there would be no electrical response. A negative electrical response at operation is thus of little value. Longitudinal incision and partial consecutive incisions, as well as palpation, may give additional information concerning the relative proportion of funiculi and scar tissue.

There are three main types of operations on injured nerves: Neurolysis or nerve liberation; nerve suture, complete or partial; nerve transplantation.

Nerve liberation consists in freeing the nerve trunk from all surrounding scar and callus. In many instances the nerve may be found completely embedded in scar tissue and the nerve trunk itself invaded. Nerve liberation is successful only when the nerve trunk has been completely freed and when there is no scar within the nerve trunk. In the presence of interstitial changes, excision and nerve suture must be undertaken. In instances in which there is slight interstitial scar tissue, conservative measures may be attempted. In such cases by injecting salt solution, under pressure, into the nerve, adhesions may be broken up and new channels opened for the neuraxes, since we know that neuraxes may grow through loosened scar tissue. This procedure when successful gives early and most rapid return of function and, therefore, is well worth trying in properly selected cases. These cases constitute the major portion of nerve injuries which recover without operation. And even in many of these, recovery is greatly hastened by this simple operative procedure. If marked evidences of recovery do not take place within a few weeks, it is evident that there has been an error in judgment at the time of operation, and a more radical procedure must be undertaken. However, if these possibilities are borne in mind, little time is lost and much may be gained. Hence the procedure may well be essayed.

The question of tubulization of a neurolyzed nerve deserves consideration. It has been suggested that the nerve should be surrounded loosely by a pedicle of fat which is carried beneath the nerve and carried back over the superior surface and anchored. The pedicle should be larger than the area to be covered to allow for subsequent shrinkage and should be selected with consideration as to blood supply. Opinions are not unanimous as to the value of tubulization with a sheath of fat completely severed from its blood supply. The persistence, for a period of weeks, of a free fatty sheath in scar tissue has not been fully established. Recently Moynihan has advised against the use of fat, claiming that it interferes with vascu-

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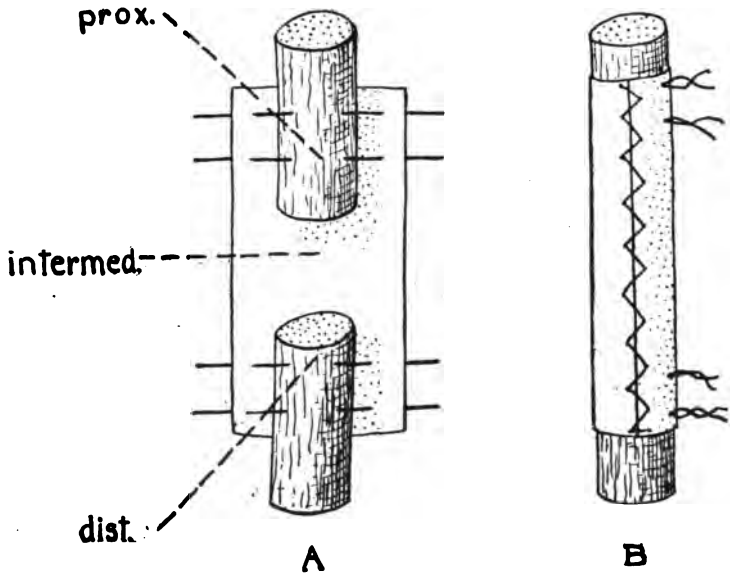

FIG. 159.—Illustrating fascial tubulization. (Kirk and Lewis.)

If without undue liberation of a nerve, proximal and distal to the scar, this can be placed in a new bed in neighboring muscle tissue, thus avoiding the scar bed, such procedure is deserving of consideration.

Surgical intervention may be indicated in spite of evidence of progressive nerve regeneration in cases in which there is exquisite and intractable pain. The exact pathology of causalgia is not fully understood. However, some cases are caused by pressure on the nerve from without and by scar formation within the nerve. In the former group relief may be immediate after freeing the nerve from scar, callus, or from around bony exostoses. When the cause lies within the nerve Sicard has recommended the injection of 60 per cent alcohol. It should be understood that this method leads to partial or complete degeneration of the distal part of the nerve fol-

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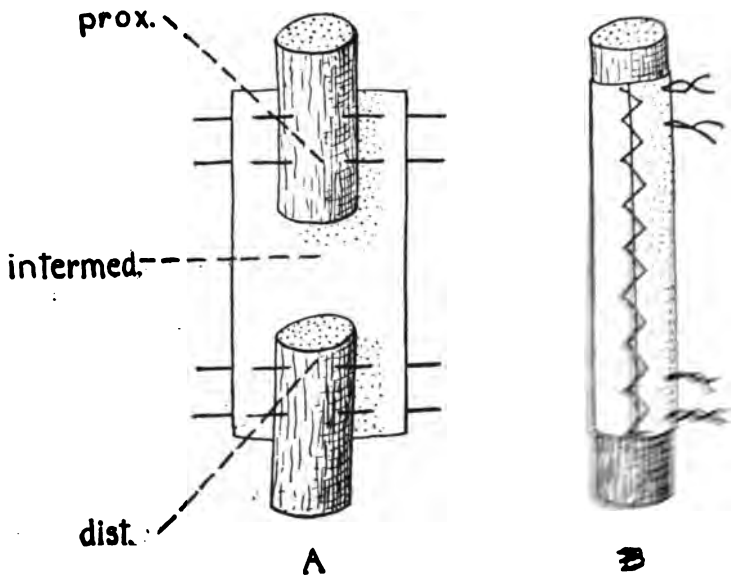

FIG. 159.—Illustrating fascial tubulization. (Kirk[illegible])

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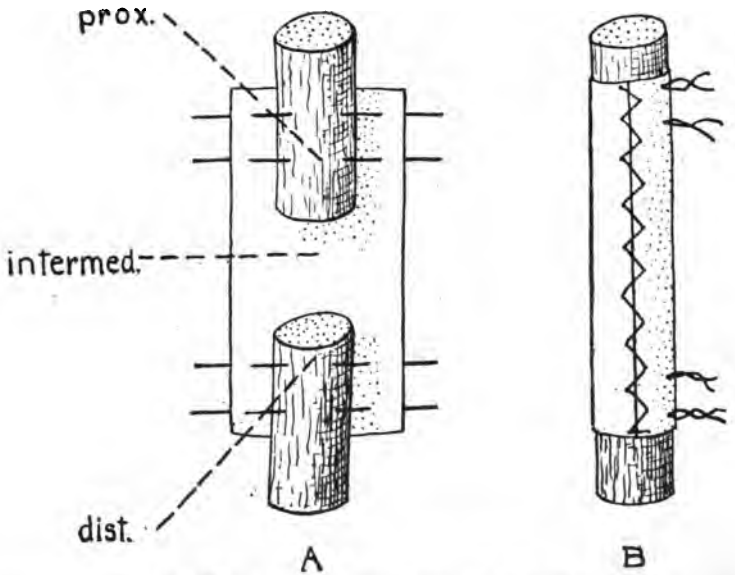

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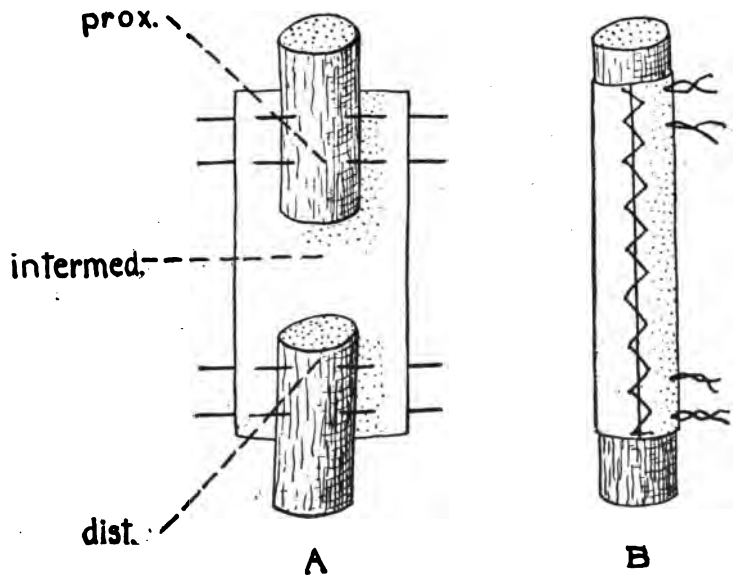


FIG. 159.—Illustrating fascial tubulization. (Kirk and Lewis.)

If without undue liberation of a nerve, proximal and distal to the scar, this can be placed in a new bed in neighboring muscle tissue, thus avoiding the scar bed, such procedure is deserving of consideration.

Surgical intervention may be indicated in spite of evidence of progressive nerve regeneration in cases in which there is exquisite and intractable pain. The exact pathology of causalgia is not fully understood. However, some cases are caused by pressure on the nerve from without and by scar formation within the nerve. In the former group relief may be immediate after freeing the nerve from scar, callus, or from around bony exostoses. When the cause lies within the nerve Sicard has recommended the injection of 60 per cent alcohol. It should be understood that this method leads to partial or complete degeneration of the distal part of the nerve fol-

lowed by retarded regeneration. Simple section of the nerve, central to the region of the scar, therefore, may be considered in such cases.

Direct nerve suture should be done only when the nerve ends can be brought in apposition without tension. Gentle traction may be used after the manner already described, care being taken to avoid too strenuous a pull, since by so doing, karyolysis of the motor cells in the anterior horn takes place, with subsequent degeneration of the neuraxes within the proximal stump—a result studiously to be avoided. Defects in the nerve may be lessened by altering the position of the extremity. The position varies with the nerve and the level of the lesion. By means, then, of stretching and altering the position of the limb a few centimeters may be gained, two to three in the upper arm and three to four in the thigh. The nerve ends must not be sutured under tension. If this is done with the limb in the overcorrected position, there would then result, when the limb is gradually lowered, an elongation and an increase of the scar which probably would prevent the down-growth of the neuraxes.

When excising dense scar within the nerve trunk, it is imperative to excise liberally. There should be little or no regard concerning the amount removed. Before suturing a completely severed nerve, the neuroma present on the central stump must be resected until normal nerve tissue is reached and the central end of the distal stump liberated and likewise resected until its component funiculi are distinctly evident. Great caution should be observed not to remove tissue needlessly. This is best safeguarded by making relatively thin consecutive sections incompletely through the scar or neuroma. In this manner one can more readily recognize approaching normal nerve funiculi.

Partial nerve suture is rather frequent in injuries of the larger nerves, more particularly the peroneal portion of the sciatic. The aim here is to remove all interstitial scar, without section of the normal nerve funiculi. The incisions should be sharp and clean cut, avoiding all tearing of the nerve. The same suture is to be used and the same technic as in complete end-to-end suture.

When the nerve defects are so extensive that the severed ends can not be brought together without undue tension, as is unfortunately frequently the case, methods of bridging the defect or other operative procedures should be considered.

In experimental work perfect anatomical repair of an inch or a little more can be secured by tubulization with fascia. After tubulization with fascia, the fascial tube which has been formed should not be placed in a bed of scar tissue, but should be placed in adjacent muscle or subcutaneously in the fat.

In most cases, however, the operation of choice is that of *nerve transplantation*, since a transplanted nerve segment offers the most

favorable path for the down-growth of central neuraxes. Whenever possible, auto-transplants should be employed. The nerves used are the radial, just below the elbow and the musculo-cutaneous of either the arm or the leg and the sural. Since these nerves are, as a rule, of much smaller diameter than the severed nerve to be bridged, several segments of the selected nerve are to be used. Each segment is sutured in place separately, both at the proximal and the distal ends, employing essentially the same technic as in simple suture. Great care should be exercised so as to obtain end-to-end approximation of the several nerve segments transplanted and the severed ends of the nerve to be bridged. Experimentally it has been shown that three or four segments of a small nerve thus transplanted, while retaining their identity, are early bound together by newly formed connective tissue to form one bundle. Several segments of a small nerve thus bound together give opportunity for the down-growth of a much greater number of neuraxes growing from the central stump, and also from the different regions of the same.

Homo-transplants, removed, for instance, after amputations, may be used when available within a short time after amputations. If the nerve segment to be transplanted is of essentially the same size as that resected the technic of suture, proximal and distal, is the same as that given under simple suture. It is probable that better results are to be expected if several segments of a small nerve are transplanted as described under multiple auto-transplants.

The question of tubulization after nerve-grafting has not received final answer. In general, the same considerations maintain as discussed under neurolysis.

None of the methods of tubulization without nerve transplantation, using Cargile membrane, hardened arteries, veins, gelatine tubes, tubes filled with agar, have been reported with a sufficient number of end results to admit of forming definite conclusions. Experimental evidence admits of the conclusion that methods of tubulization are not as satisfactory as nerve transplantation.

Various other methods have been suggested to bridge the gap between the ends of a severed peripheral nerve in cases where direct suture can not be performed. Of these methods mention may be made of suture at a distance with catgut; nerve flaps taken either from the central or the distal stump or both; nerve anastomosis or grafting. (Fig. 160.) All of these methods have little regard for the fundamental principles of nerve regeneration, and have proven unsatisfactory on experimental test. They should then be condemned as operative procedures.

Hofmeister has recently reported operations of multiple grafting in cases in which a number of nerves of an extremity have been injured and segments of considerable length destroyed. Enough

time had not elapsed in any of the cases to admit of a statement being made concerning ultimate results.

Judging from results obtained in experimental observations on simple nerve grafting it may be stated that this operation does not commend itself as offering favorable opportunity for the regeneration of the distal segments of the nerves thus engrafted.

Shortening the limb by resection of the bone is never justifiable in cases where nerve defects are very extensive. Usually some form

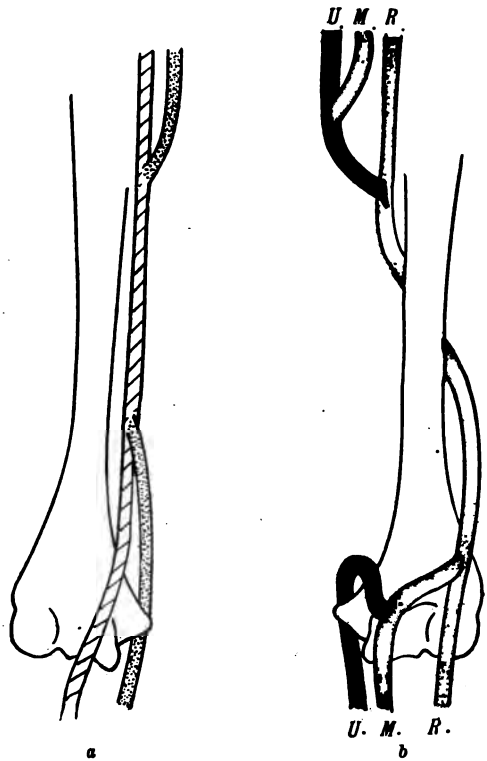


FIG. 160—(*a* and *b*).—Illustrating methods of performing nerve implantation.

a. Illustrating method of implanting ulnar, a long segment of which has been destroyed, into median. Ulnar nerve indicated by stipple, and median by oblique lines.

b. Illustrating method devised by Hofmeister in which long segments of adjoining nerves have been destroyed and an attempt is made to carry their fibers to the periphery through another nerve. U, ulnar nerve. M, median. R, radial.

of plastic operation can be done which would give better end-results. In certain particular nerve injuries, when taken in consideration with the injury complex as a whole, it may be advisable to perform a tendon transplantation rather than some form of nerve bridging. This has been discussed by Col. Sir Robert Jones.

Complications.—The most frequent complications met with in operations for the repair of peripheral nerves are injuries to blood vessels, including thrombosis and aneurysm. In not a few instances the nerves are bound down in a mass of scar tissue and intimately adherent to the great vessels. Aneurysms are particularly found in injuries to the plexuses, cervical and brachial, and also to the nerve trunks in the axilla. Such complications can usually be recognized before operation. Ocasionally they are overlooked and are masked by the nerve injury. Unless borne in mind, at the time of operation, they may lead one into extreme operative difficulties. Aneurysm should be suspected when there is extreme atrophy together with considerable fibrous change in the muscle. The picture borders onto that of Volkman's contracture. If the cardinal signs of traumatic aneurysm are looked for they can usually be elicited. The combined nerve and arterial lesion is difficult to handle and offers a poor outlook.

Another complication, occasionally found, is injury to the apex of the lung. At the time of injury this is overlooked, due to the extreme paralysis as a result of the lesion of the plexus. In cleaning out the scar tissue and in freeing the nerve trunks at the operation for nerve repair, the pleural cavity may be again opened. Such a complication should always be thought of in injuries to the lower cervical trunks.

Prognosis.—According to the statistics of Tinel, between 60 and 70 per cent of cases of nerve injury recover without any surgical intervention. According to the same author, in 108 cases of nerve suture or nerve grafting, there have been 22 cases of practically complete restoration, 14 complete failures, and all the rest of the cases had improved or were still improving. From Marie's Clinic Mme. Athanassio-Bénisty reports:

Liberation of nerve: 50 cases; about 20 complete recoveries, 1 failure, the rest improved.

Cases where there was no division of the nerve, but some swelling. The nerve was freed and several longitudinal slits made in the smaller portion: 50 to 60 cases; 25 cures, 1 failure, 30 improved.

Suture: 11 cases; 2 complete cures, 3 improvements, 6 cases slight or no improvement.

Resection followed by sutures: 5 cases; 2 almost complete cures, 1 partial, and 2 failures.

Partial resections: 4 cases; 2 very satisfactory results, 2 failures. Nerve grafts; 2 cases; after 1 year 8 months show only the slightest signs of recovery.

The best results were obtained with the radial and external popliteal, not so good with the ulnar, and least satisfactory with median

The musculo-spiral nerve.—The musculo-spiral nerve is the most frequently injured of all the nerves of the body. It arises from the posterior cord of the brachial plexus and bends around the back of the humerus where it lies under cover of the triceps muscle. It appears on the lateral surface of the humerus and then on the anterolateral surface of the musculo-spiral groove. From here it descends between the supinator longus and the biceps muscle to the elbow. Just below the elbow it divides into its terminal branches, the radial

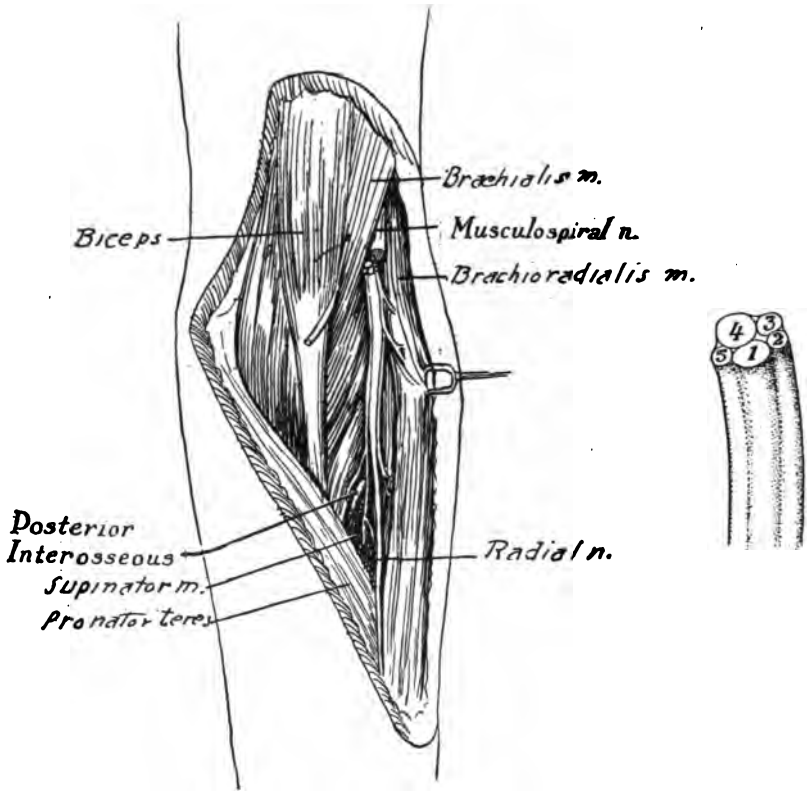


FIG. 163.—Internal topography of the musculospiral (N. Radialis) in the elbow. The nerve is cut through and the distal end turned up. On the side is shown a cross section of the distal stump, bringing out the different tracts. 1. Radial nerve. 2. Tract for the brachioradialis. 3. Tract for the extensores carpi radialis. 4. Tract for the ramus profundus (exten. digit. comm., exten. pollicis and exten. radicis.). 5. Tract for the supinator longus. (Vulpius and Stoffel.)

and the posterior interosseous. The radial is a purely sensory nerve. It descends under cover of the supinator longus and becomes subcutaneous in the lower part of the forearm, to be distributed to the dorsum of the hand. The posterior interosseous passes around the head of the radius and descends in the forearm between the deep and superficial muscles, supplying all the muscles of the forearm.

Motor.—In the upper part of the arm the musculo-spiral supplies the triceps and a branch to the anconeus.

In the bicipital groove in the lower part of the arm, it supplies the supinator longus and the extensor carpi radialis longior. As it passes around the head of the radius it gives off branches to the extensor carpi radialis brevior and the supinator brevis. In its course through the forearm it supplied all the muscles on the back of the forearm.

Sensory.—There are three sensory branches: (1) A small branch given off high up in the upper arm to the postero-internal portion of the upper arm; (2) the external cutaneous branch, given off in the middle of the arm and supplying a narrow strip of skin on the postero-external surface of the forearm; (3) the radial nerve, one of the terminal branches, supplies the dorsum of the hand, and on the radial side, extending up into the fingers and thumb, as far as the proximal inter-phalangeal joint.

FIG. 164.—Sensory loss accompanying a lesion of the musculo spiral nerve before the external cutaneous branches had been given off. To pin-pricks there was blunting over the shaded area. There was no epicritic loss.

Paralysis of the musculo-spiral results in the following symptoms:

Motor.—1. Inability to extend the elbow (triceps).

2. Inability to extend the wrist (extensor carpi radialis longior and brevior and extensor carpi ulnaris).

3. Inability to extend the fingers (extensor communis digitorum).

4. Inability to extend the thumb (long and short extensor of the thumb and extensor ossis metacarpi pollicis).

5. Flexion of the elbow is weakened as a result of the paralysis of the supinator longus. The supinator longus is a powerful flexor of the elbow, acting especially when the hand is midway between pronation and supination.

6. Weakening of supination (supinator longus and brevis).

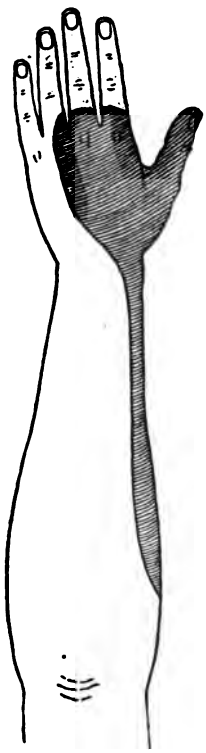


FIG. 165.—Musculo-spiral paralysis, with blunting of sensation to pin-prick and cotton-wool over the shaded area.

The striking feature of paralysis of the musculo-spiral, because it is so evident on simple inspection, is *wrist-drop*. In these cases there is some weakness of flexion of the fingers as seen in the attempt to close the wrist. This is due not to any actual weakness of the muscles of flexion, but to the absence of the synergic action of their

antagonists (the extensors). In order to have an efficient closing of the fingers, the hand must be supported in partial hyper-extension by the action of the extensors of the wrist. This action is absent in the musculo-cutaneous and internal cutaneous suffices to prevent any hyper-extended position, closure of the hand becomes firm.

Sensory.—The small sensory branch in the upper arm has no exclusive sensory supply. Complete division of the nerve results in loss of cutaneous sensibility over the dorsal aspect of the hand (radial side) and thumb, and part of the proximal phalanges of the index and middle fingers. Occasionally there is some loss of sensation on the dorsum of the forearm, but usually the overlap from the musculo-cutaneous and internal cutaneous suffices to prevent any definite zone of anesthesia in this region.

FIG. 166.—Complete interruption of the musculo-spiral, nine months previously. Crushing of the nerve and involvement in callus of fractured humerus. Extreme hypotonia.

Any lesion above the point where the external cutaneous branch of the musculo-spiral is given off will give this anesthesia over the dorsum of the hand.

A lesion below the point of origin of the external cutaneous branch results in no loss of sensation. This holds good until we reach the lower forearm and wrist. At this level, branches from the external cutaneous and from the radial anastomose. And from here on down, division of the nerve will produce loss of cutaneous sensibility over the dorsum of the hand as described above. The zone of anesthesia is more extensive from a lesion at the wrist than from one in the lower forearm.

1. *Complete paralysis* then of the musculo-spiral will give paralysis of extension of the elbow, wrist-drop (Fig. 166), weakness of supination and flexion of the forearm, and a sensory loss in the

dorsum of the hand. The triceps reflex will be absent. To do this, the lesion must affect the nerve high up, as occurs in cases of crutch-palsy or the so-called Saturday night palsy.

2. *Injury above the point of origin of the nerve to the supinator longus.*—This is the most common site of injury to the nerve. It may result from fracture of the humerus or from the callus following a fracture, although the former is more frequent. It will give the same picture as that described above excepting that the triceps and the triceps reflex are spared. The external cutaneous branch may or may not be affected. If it is spared there will be no sensory loss.

3. *Below the supinator longus nerve.*—This will give essentially the picture of a wrist-drop. The nerve to the extensor carpi radialis longior arises just below that to the supinator longus and may also be spared in this type of lesion, either in whole or in part. If it is spared, some extension of the wrist will be possible on the radial side. That is, there will be some extension of the wrist associated with abduction. There will be no sensory loss.

4. *In the forearm.*—The radial and the posterior interosseous nerves separate at the elbow. A little below the elbow a wound of the posterior interosseous will spare the nerve to the short radial extensor. In that case extension of the wrist on the radial side will be intact. There will be paralysis of the ulnar extensor of the wrist, of the extensors of the fingers, and of the extensors of the thumb. No sensory loss will occur.

Injuries to the posterior interosseous below this point may catch isolated nerves to one or the other of the muscles of the fingers and thumb.

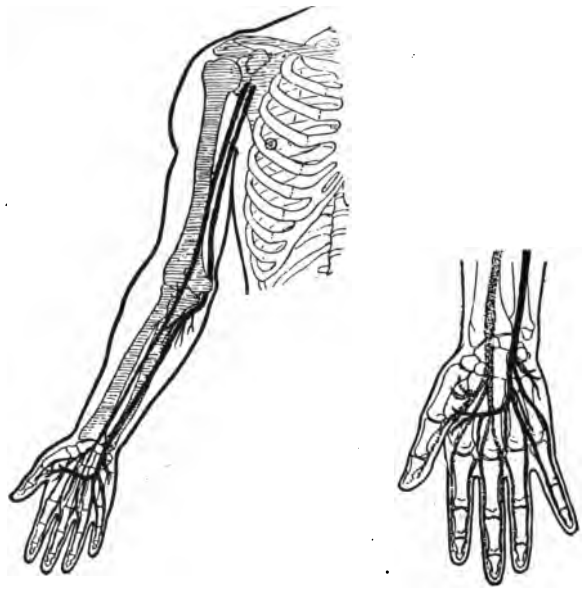
Injury to the radial nerve in the upper forearm will give no sensory loss. Injury in the lower one-third of the forearm, and from there on down, will cause a zone of anesthesia as described above.

Dissociated syndromes.—Where the nerve lies on the antero-external surface of the humerus the branches to the supinator longus and to the extensor carpi radialis longior seem to lie in the postero-external portion of the cross section of the trunk. Partial lesion of this portion of the trunk may affect only these two muscles. As far as known, the general topography of the cross section of the musculo-spiral nerve in this region is from front to back; laterally, radial, supinator longus, radial extensors; mesially, ulnar extensor of the wrist and the extensors of the thumb and fingers, and the supinator brevis.

Symptoms of nerve irritation are frequent in musculo-spiral palsy.

In treatment of musculo-spiral palsy it is important to remember that the hand must be kept in hyper-extension.

. **Ulnar nerve.**—The ulnar nerve arises from the inner cord of the brachial plexus along with the inner head of the median. In the upper arm it lies on the inner side of the brachial artery and close to the median. In the lower part of the arm it bends away from the artery and passes to the back of the humerus, where it lies in the groove between the internal condyle of the humerus and the olecranon process. In the forearm it passes through the flexor carpi ulnaris and runs beneath this muscle to the wrist. Here it pierces the deep fascia and enters the hand. At this level it gives off its two terminal branches, the superficial palmar branch, which is sensory, and the deep palmar branch, which is motor.



FIGS. 167 and 168.—Course of ulnar and median (diagrammatic).

Motor.—In the forearm the ulnar nerve supplies the flexor carpi ulnaris, and the inner portion of the flexor profundus digitorum (destined for the ring and little fingers). In the hand it supplies the muscles of the hypothenar eminence, all the interrossei, the two inner lumbricals, and in the thenar eminence the adductor pollicis and the short head of the flexor brevis pollicis.

Sensory.—The sensory supply is exclusively to the hand. The superficial palmar branch supplies the skin over the inner part of the palm, as far as the wrist, the little finger and one-half the ring finger. The dorsal palmar branch, which is given off from the nerve about the middle of the forearm, supplies the inner half of the dorsum of the hand as far as the wrist, the little finger and one half the ring finger.

I. *Complete paralysis of the ulnar—Motion.*—Flexion of the wrist is impaired, and in flexion, the hand is tilted to the radial side, due to loss of the flexor carpi ulnaris. This loss is fairly compensated by the palmaris longus. Paralysis of the muscles may be detected by failure to feel the tendon contract at its point of insertion at the wrist.

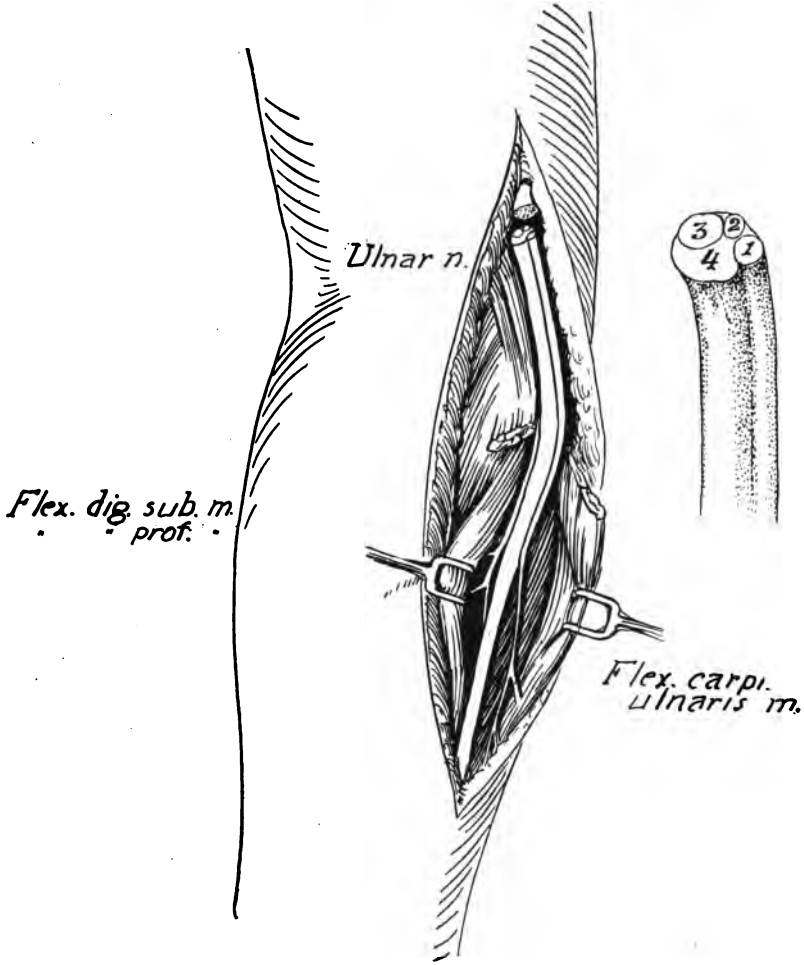


FIG. 169.—A. Humeral head of the flexor carpi ulnaris is freed from the bone, the flexor sublimis digitorum drawn aside. To demonstrate the relationships of the cross section of the ulnar nerve, the nerve is shown cut through. B. Peripheral stump of the ulnar nerve, showing the different tracts. 1. Tract for flexor carpi ulnaris. 2. Tract for flexor profundus digitorum. 3. Tract for the ramus profundus (muscles of the hypothenar eminence, interossei, third and fourth lumbricals, adductor pollicis and deep head of the flexor brevis pollicis. 4. Sensory tract. (Vulpius and Stoffel.)

Flexion of the little and ring fingers is greatly impaired. Some flexion is possible through the flexor sublimis digitorum, supplied by the median. But flexion of the terminal phalanges is impossible, ow-

ing to paralysis of the portion of the flexor profundus digitorum destined for these fingers.

Interossei: The normal function of the interossei is adduction and abduction of the fingers, flexion of the fingers at the metacarpophalangeal joints, and extension at the interphalangeal joints.

Abduction and adduction of the fingers is lost. This should be tested on a flat surface, to avoid any confusion with the long flexors and extensors, which also have some adduction and abduction action.

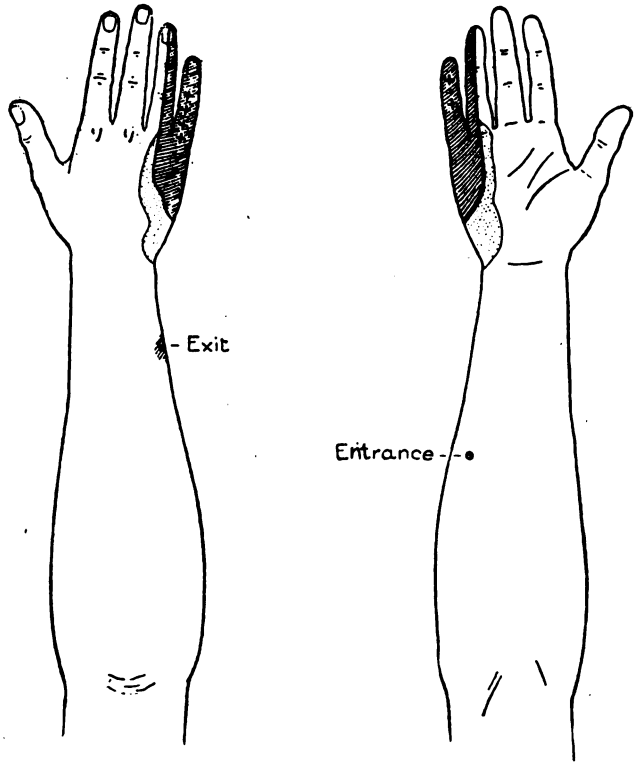


FIG. 170.—Cutaneous supply of the ulnar nerve. The shaded area is that insensitive to pin-prick (protopathic loss). The dotted area is the additional area insensitive to cotton-wool touches (epicritic loss). The crosses indicate the region where there is loss of sensation to deep pressure. Joint sense is lost in the little finger and vibration-sense in the two ulnar fingers. The ulnar nerve and its dorsal cutaneous branch were embedded in dense scar tissue.

There is hyperextension of the fingers at the metacarpophalangeal joints, due to loss of the interossei (which flex these joints) and the unopposed action of the long extensors of the fingers. There is flexion at the interphalangeal joints, due also to paralysis of the interossei (which extend them) and the unopposed action of the flexors. This hyperextension of the matacarpophalangeal joints, and flexion of the interphalangeal joints, are both more marked for

the little and ring fingers than for the middle and index fingers. This is due to the sparing of the two ~~inner~~ lumbricals, which are supplied by the median. The above picture gives the characteristic ulnar deformity.

Adduction of the thumb is lost, through paralysis of the adductor pollicis. This movement also should be tested with the hand on a flat surface, to prevent confusion with a somewhat similar action by the long flexor of the thumb.

Sensory.—There is epicritic loss over the sensory area described above. The area of epicritic loss for the ulnar has very sharp and

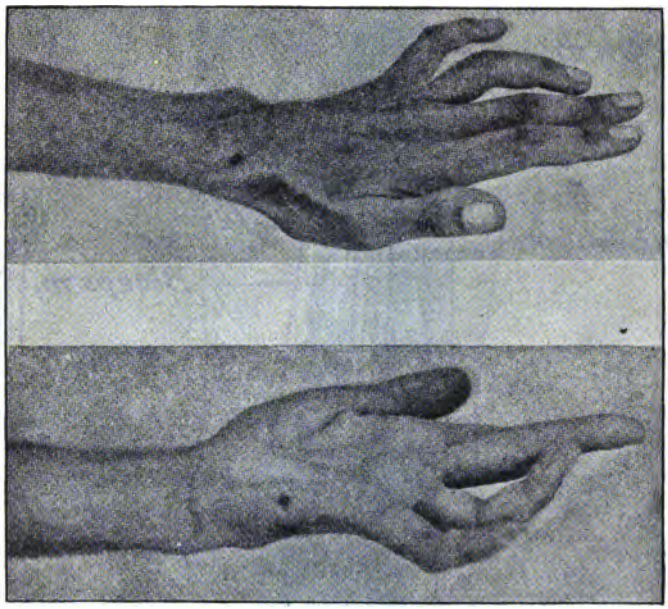


FIG. 171.—Bullet wound of the deep branch of the ulnar nerve accompanied by no sensory loss, and with joint-sense normal in all joints. Entrance-wound at ulnar border of hand, 2 inches below the wrist. Exit-wound on dorsum, the bullet having traversed the carpus. Note the position of the two ulnar fingers: hypo-extension at the metacarpo-phalangeal joints and semi-flexion at the interphalangeal joints.

well-defined borders. The protopathic loss is considerably smaller and less constant.

II. *Injury below the elbow.*—The symptoms will be the same, except for preservation of ulnar flexion of the wrist, and flexion of the terminal phalanges of the little and ring fingers.

III. *Injury at the wrist.*—The dorsal cutaneous branch having already been given off, sensory disturbance will be confined to the palm.

Dissociated syndromes.—Some light has been obtained on the inner structure of the ulnar nerves, through occurrence of partial lesions.

In the cross section of the nerve from within outward, the following arrangement apparently occurs: (1) Cutaneous branches and branches to the hypothenar eminence; (2) fibers to the interossei; (3) fibers to the adductor pollicis; (4) fibers to the flexor carpi ulnaris and the flexor profundus digitorum.

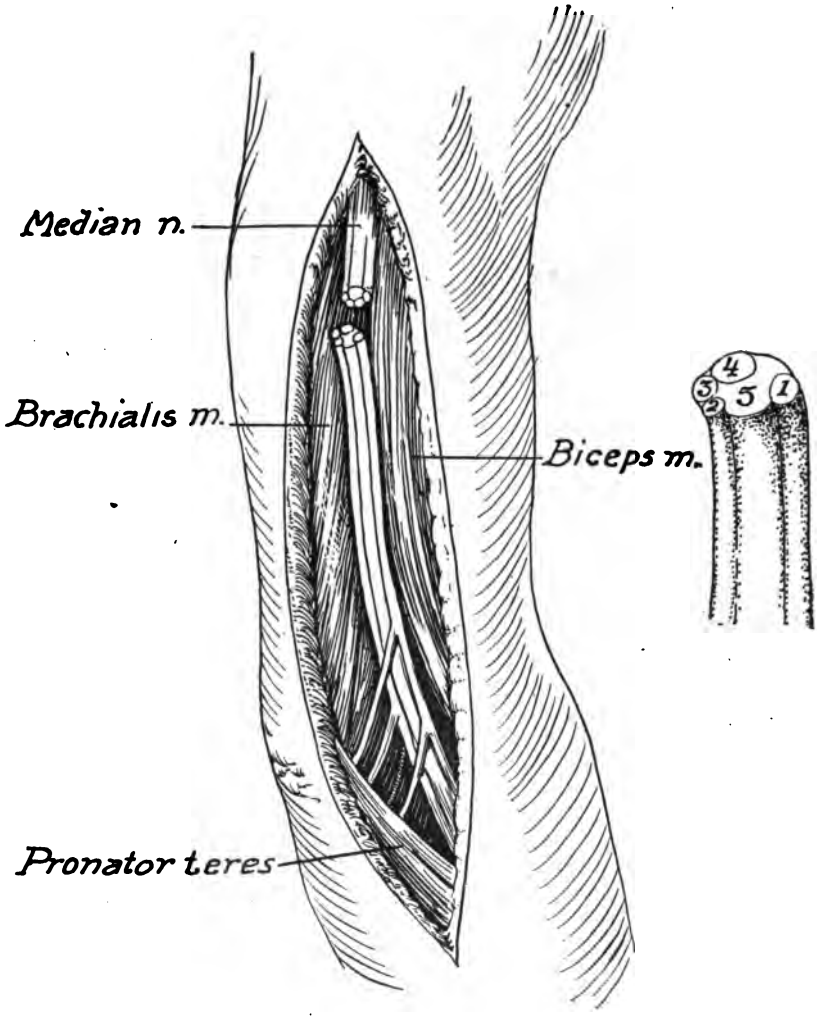


FIG. 172.—Location of nerve tracts in the median. Topography of the cross section. The nerve is shown cut through in the upper arm, and turned back. The enlarged cross section shows the arrangement of the nerve tracts as follows: 1. Tract for the pronator radii teres, flexor carpi radialis and palmaris longus. 2 and 3. Tracts for the flexor sublimis digitorum. 4. Tract for the flexor profundus digitorum and flexor longus pollicis. 5. Sensory tracts, and tracts to the muscles of the thenar eminence and lumbricals (first and second). (Vulpius and Stoffel.)

Irritations of the ulnar nerve are especially apt to lead to muscular hypertonia, contraction, and even deformities. It is especially in association with ulnar lesions that the anomalous "reflex contrac-

tures" of Babinski occur. On the other hand, causalgias are not common from ulnar lesions.

Median nerve.—The median arises by two heads, one from the outer and one from the inner cord of the plexus. The nerve descends along the lateral surface of the brachial artery, crossing it in the lower half of the arm. At the bend of the elbow it lies mesial to the

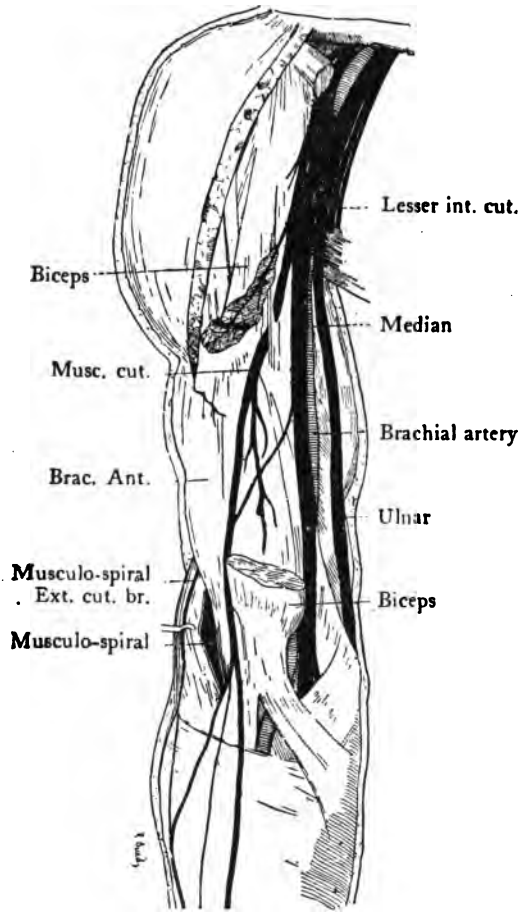


FIG. 173.—Deep nerves of the arm (after Hirschfeld) modified. Anterior aspect.

artery. Entering the forearm between the heads of the pronator radii teres, it runs down the forearm between the superficial and deep muscles, gradually shifting to the radial side. At the wrist it becomes almost subcutaneous, lying just beneath the tendon of the palmaris longus. (Fig. 173.)

Motor.—The median supplies all the muscles of the anterior surface of the forearm except the flexor carpi ulnaris and inner por-

tion of the flexor profundus digitorum; that is, pronator radii teres, flexor carpi radialis, palmaris longus, flexor sublimis digitorum, outer heads of the flexor profundus digitorum and pronator quadratus.

In the hand it supplies the muscles of the thenar eminence except the deep head of the flexor brevis pollicis; that is, the abductor pollicis brevis, opponens pollicis, and superficial head of the flexor brevis pollicis. It also supplies the two inner lumbricals.

Sensory.—The sensory supply is exclusively to the hand. On the palmar surface, the radial half of the ring finger, the middle and index fingers, the palmar surface of the thumb, and the corresponding areas of the palm. On the dorsum of the hand the distal

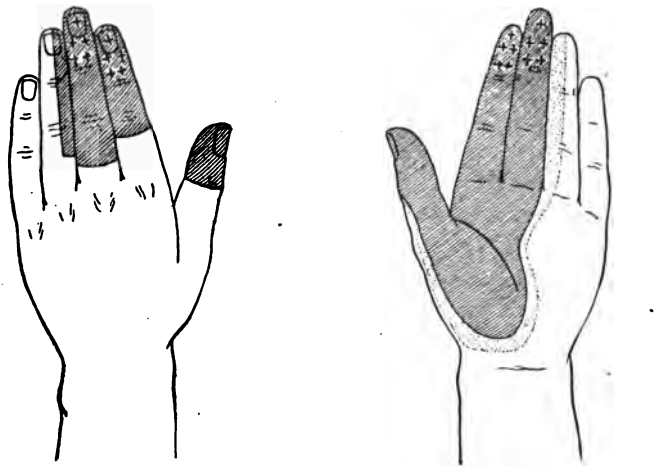


FIG. 174.—Paralysis of the median nerve. The dotted area is that insensitive to cotton-wool touches; the shaded area is that insensitive to pin-pricks and cotton-wool touches. The crosses indicate the region insensitive to deep pressure.

two and one half phalanges of the middle and index fingers, and the last two of the ring finger. (Fig. 174.)

Complete paralysis of the median—Motor.—Pronation is impossible (except passively from the weight of the arm) from loss of the pronator radii teres and pronator quadratus.

Flexion of the wrist. This movement is normally produced by the flexor carpi radialis, palmaris longus and flexor carpi ulnaris. The first two are here paralyzed. A little flexion of the wrist, toward the ulnar side is still possible.

Flexion of the fingers.—This movement is normally produced as follows: At the metacarpophalangeal joints by the interossei; at the proximal interphalangeal joint by the flexor sublimis digitorum; at the interphalangeal joint by the flexor profundus digitorum.

For the ring and little fingers the flexor profundus is unaffected (ulnar supply), so that considerable flexion is left, but weaker than normal from loss of the flexor sublimis digitorum. For the middle and index fingers, both flexor sublimis and flexor profundus digitorum being gone, only the slightest flexion from the interossei remains.

Thumb.—The terminal phalanx can not be flexed from loss of the flexor longus pollicis and superficial head of the flexor brevis pollicis. Abduction of the thumb and opposition of the thumb are lost, due to paralysis of the abductor pollicis brevis and the opponens pollicis. Substitutions by other muscles may be confusing. In abduction the thumb should come straight out in a plane at right angles to the

FIG. 175.—Paralysis of the median nerve. Complete interruption above the epitrochlea. Maximum flexion of the fingers.

palm. In opposition, the thumb rotates so that its palmar surface looks toward the palm.

Sensation.—Epicritic sensibility is lost over the area described above as supplied by this nerve. It is sharply defined and constant. Protopathic loss is much smaller and less constant. There is no loss of deep sensibility unless tendons at the wrist are divided. Because of the preservation of deep sensibility, care must be used to avoid eliciting deep sensibility when testing for epicritic. (Test with cotton.)

Injury in the lower forearm and wrist—Motor.—Only the small muscles of the thumb will be involved.

Sensory.—Disturbance as above.

After complete division of the median, there is no deformity of the hand. Many of the movements lost may be substituted by other

movements, and the disability need not be great. The loss of flexion of the terminal phalanx of the thumb, and the loss of opposition of the thumb, will give the diagnosis.

Dissociated syndromes.—From the partial lesions it would seem that the fibers for the pronator radii teres, flexor carpi radialis, flexor pollicis longus, and the muscles of the thenar eminence occupy the outer part of the nerve. The fibers for the flexors are mesial.

The very severe types of nerve irritation, especially the causalgias, are more common from involvement of the median than of any other nerve.

Median and ulnar nerves. — Combined involvement of these two nerves is very frequent because of the close association of the nerves

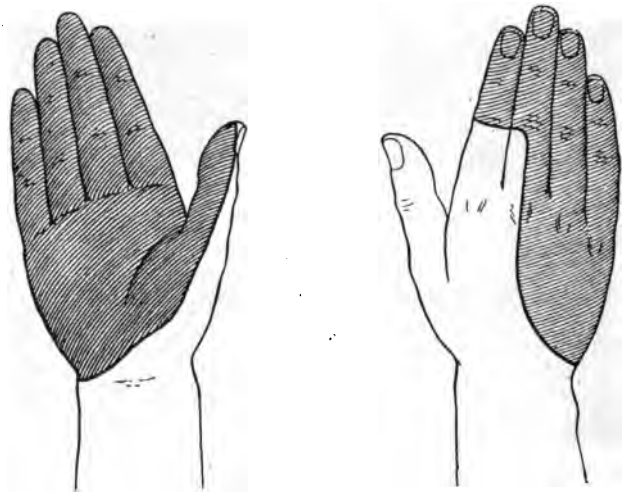


FIG. 176.—Paralysis of median and ulnar nerves. The shaded area shows the loss to pin-prick and cotton-wool. The ulnar nerve was completely divided; the median was embedded in fibrous tissue.

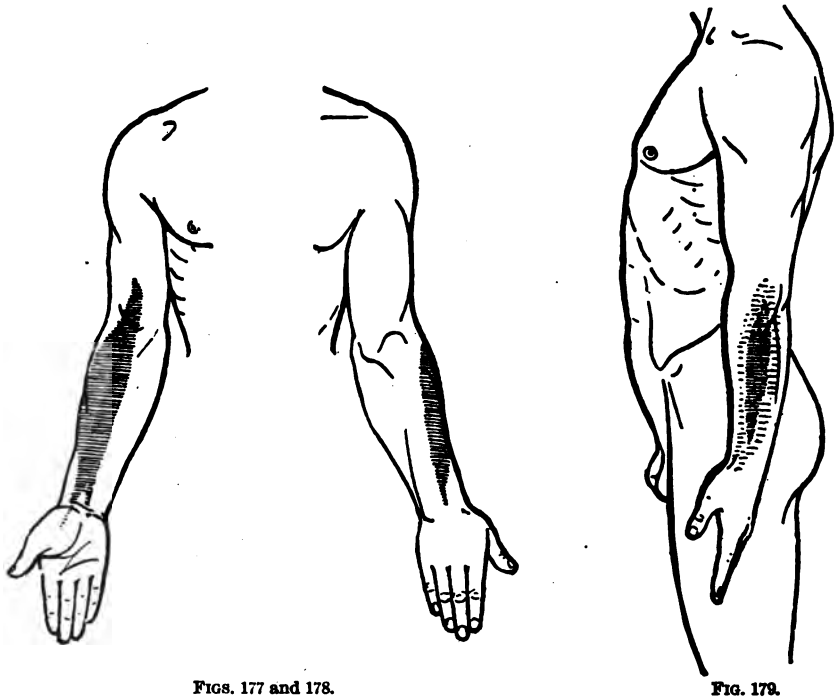
in the upper arm. The sensory loss is that of the combined sensory distribution of the two nerves but varies considerably from case to case. There is complete paralysis of all the muscles supplied by both nerves. Flexion of the wrist and fingers and pronation of the forearm are gone. All the small muscles of the thenar and hypothenar eminence and the interossei and lumbricals are completely gone. Adduction and abduction of the fingers and opposition of the thumb and fingers are lost. There is marked wasting of the palm.

Musculo-cutaneous nerve.—The musculo-cutaneous nerve arises with the outer head of the median from the outer cord of the brachial plexus. It runs with the median in the upper third of the arm, then passes laterally through the coraco-brachialis and descends between the biceps and brachialis anticus to the elbow. In the forearm it becomes the external cutaneous nerve and is purely sensory.

Motor.—Supplies the coraco-brachialis, biceps, and brachialis anticus.

Sensory.—Supplies a long strip on the antero-lateral aspect and to a lesser extent on the postero-lateral aspect of the forearm.

Injury to the musculo-cutaneous in the upper third of the arm results in paralysis of the muscles supplied which are the chief flexors of the forearm. Some flexion of the forearm, however, is still possible by means of the supinator longus. Supination of the arm is weak, from paralysis of the biceps.



FIGS. 177 and 178. FIG. 179.

FIGS. 177 and 178.—Sensory region of musculo-cutaneous nerve.
FIG. 179.—Cutaneous anesthesia in complete section of musculo-cutaneous nerve.

Sensory.—The sensory loss is ill-defined posteriorly, where there is considerable overlapping with the musculo-spiral. On the front of the forearm, where it comes in contact with the internal cutaneous, it is well defined. (Figs. 177, 178, 179.)

Internal cutaneous and lesser internal cutaneous nerves.—The internal cutaneous and lesser internal cutaneous are seldom affected alone. Usually either the median or ulnar or both suffer at the same time. They arise from the inner cord of the plexus and are purely sensory in function, supplying the inner surface of the arm and the antero-internal and postero-internal surface of the forearm. Injuries of these nerves will cause a corresponding loss of cutaneous sensation.

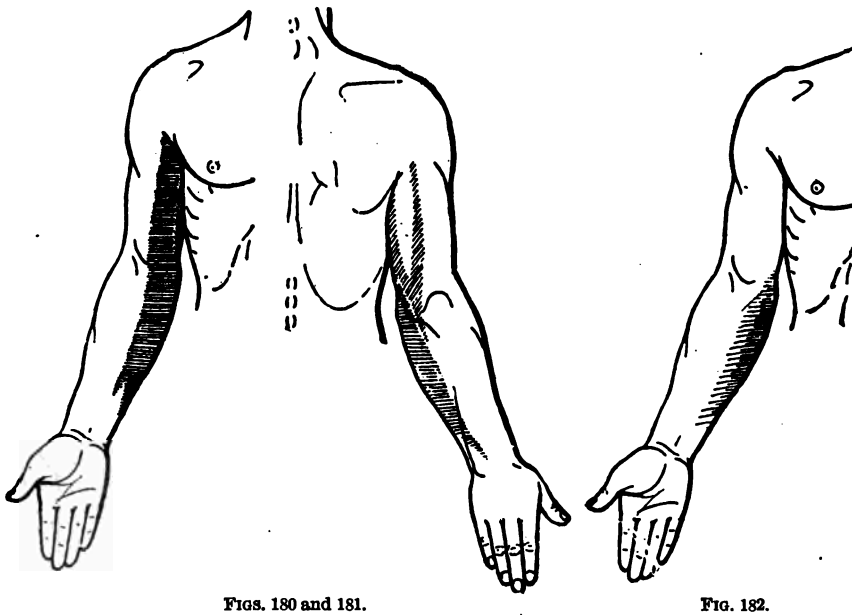
Circumflex.—The circumflex nerve arises from the posterior cord of the brachial plexus. It winds around the posterior surface of the neck of the humerus, to enter the deltoid muscle.

Motor.—Deltoid and teres minor.

Injury to the circumflex results in paralysis of the deltoid. Abduction of the arm is impossible. Very slight substitution movement is possible through the supraspinatus.

Sensory.—Supplies a skin area on the upper lateral aspect of arm and shoulder.

The loss is over the region above described.



FIGS. 180 and 181. FIG. 182.

FIGS. 180 and 181.—Cutaneous topography of the internal cutaneous and the lesser internal cutaneous (oblique hatching). The perforating branches of the second and third intercostal nerves supply a triangular area on the postero-internal surface of the arm, in the region of the lesser internal cutaneous.
FIG. 182.—Sensory disturbances in lesions of the internal cutaneous.

Isolated injury to the circumflex is exceedingly rare in war wounds. Paralysis of the deltoid is much commoner from lesions of the brachial plexus, especially of the Erb type.

Posterior thoracic.—The posterior thoracic nerve arises from the fifth, sixth, and seventh cervical roots, close to the intervertebral foramina. It passes down the side of the chest and is distributed to the serratus magnus. Paralysis of the muscle results in difficulty in bringing the arm forward to the horizontal. The attempt to do this causes the body of the scapula to push away from the chest wall.

Brachial plexus.—Injuries of the brachial plexus are among the commonest of all nerve lesions during the war, being produced directly

or indirectly by wounds of the neck, clavicle, and shoulder. Extensive wounds of the shoulder girdle often result in the tearing of muscles and tendons, so that it is difficult to distinguish those parts of the injury which are due to nerve lesions proper. Complete involvement of the plexus occurs chiefly in dislocations of the shoulder and attempts to reduce it. In wounds, several cords may be affected together, but more often a single cord or part of a cord is involved. In addition to the original trauma, scar-tissue formation and callus may lead to additional subsequent sypmtoms. Where the artery is injured ischemic paralysis may result.

The brachial plexus is formed by the anterior primary divisions of the fifth, sixth, seventh, and eighth cervical nerves, and the first

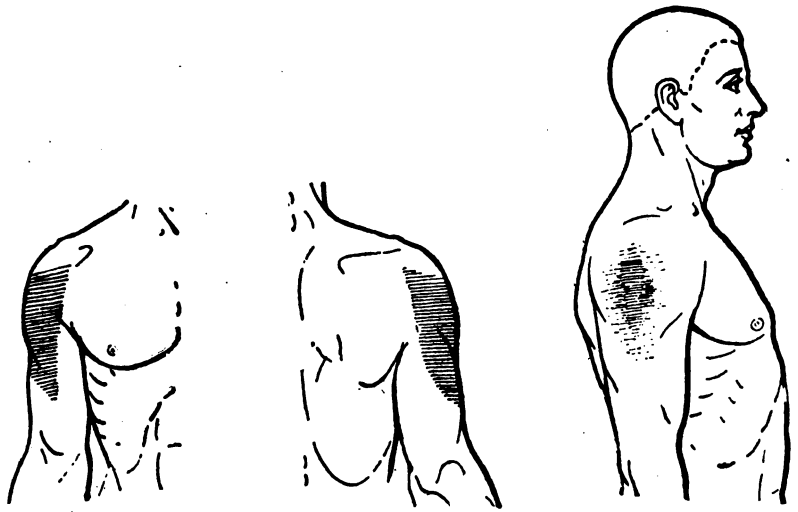


FIG. 183 and 184.—Anatomical sensory topography of the circumflex nerve (cutaneous nerve of the shoulder).

dorsal nerve. These nerves appear in the posterior triangle of the neck. After entering the posterior triangle, the fifth and sixth roots unite, the seventh remains alone, and the eighth cervical and first dorsal unite to form the three primary cords. Each of these cords then divides into an anterior and posterior portion. The anterior divisions of the fifth, sixth, and seventh cervical form the outer cord, the anterior divisions of the eighth cervical and first dorsal unite to form the inner cord, and the posterior divisions combine to form the posterior cord. The cords then break up into the nerves of distribution.

Certain nerves are given off high up in the plexus, involvement of which in the injury may aid in the localization of the lesion. These are the nerve to the rhomboids, and the long thoracic to the serratus magnus, from the fifth and sixth roots.

According to the localization of the lesion we shall get:

A. *Root symptoms* from involvement of the nerve roots or primary cords. The symptoms, both motor and sensory, will follow the laws of segmental distribution.

B. *Plexus symptoms* from involvement of the secondary cords. These have the same type of distribution, sensory and motor, as do the peripheral nerves, but differ in the way the nerves are grouped. For instance, a lesion of the inner cord will destroy the ulna and *part* (inner head) of the median.

A. Lesions of the roots and primary cords take two characteristic forms: (1) Of the upper cord (Erb-Duchenne or upper arm type): (2) Of the lower cord (Klumpke or lower arm type).

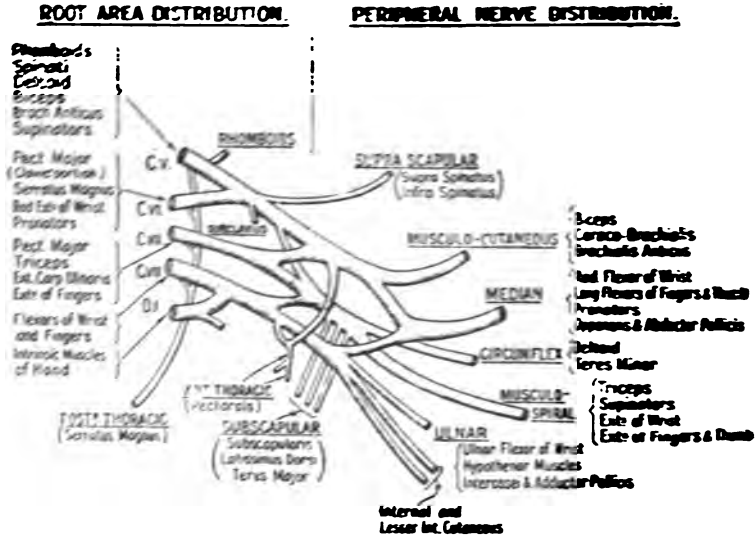


Fig. 155.—The *motor* supply of the brachial plexus, showing both its root-area distribution and its peripheral nerve distribution.

1. The *Erb-Duchenne syndrome* results essentially from a lesion of the anterior primary division of the fifth cervical nerve. Additional involvement of the sixth cervical seems to have a little effect on the extent of the paralysis. The muscles involved are those seen paralyzed in the so-called birth palsies; deltoid, biceps, brachialis anticus, and supinator longus, and in addition, some involvement of the rhomboids, spinati and sometimes of the pectorals. There is a more or less well-defined area of hypesthesia or anesthesia over the sensory distribution of the fifth and sixth cervical roots. (See Fig. 126 of sensory root areas.) Owing to the overlap of adjacent roots, however, this is variable in extent and is often wholly absent.

2. The *lower arm type* (*Klumpke*) results from a lesion of the ical and first dorsal roots or of their combined primary

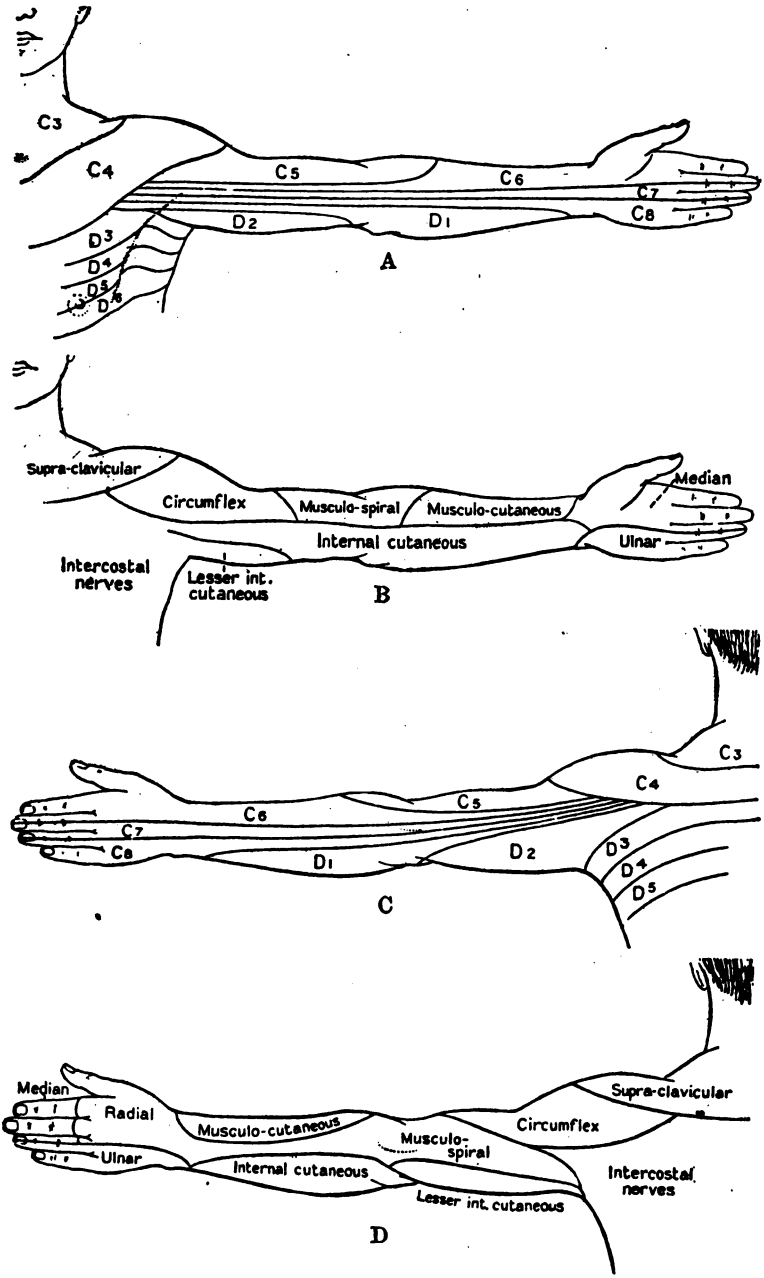


FIG. 186.—The cutaneous sensory supply of the upper limb. A and C show the areas supplied by the posterior roots; B and D the areas supplied by the peripheral nerves.

According to the localization of the lesion we shall get:

A. *Root symptoms* from involvement of the nerve roots or primary cords. The symptoms, both motor and sensory, will follow the laws of segmental distribution.

B. *Plexus symptom* from involvement of the secondary cords. These have the same type of distribution, sensory and motor, as do the peripheral nerves, but differ in the way the nerves are grouped. For instance, a lesion of the inner cord will destroy the ulna and *part* (inner head) of the median.

A. Lesions of the roots and primary cords take two characteristic forms: (1) Of the upper cord (Erb-Duchenne or upper arm type); (2) Of the lower cord (Klumpke or lower arm type).

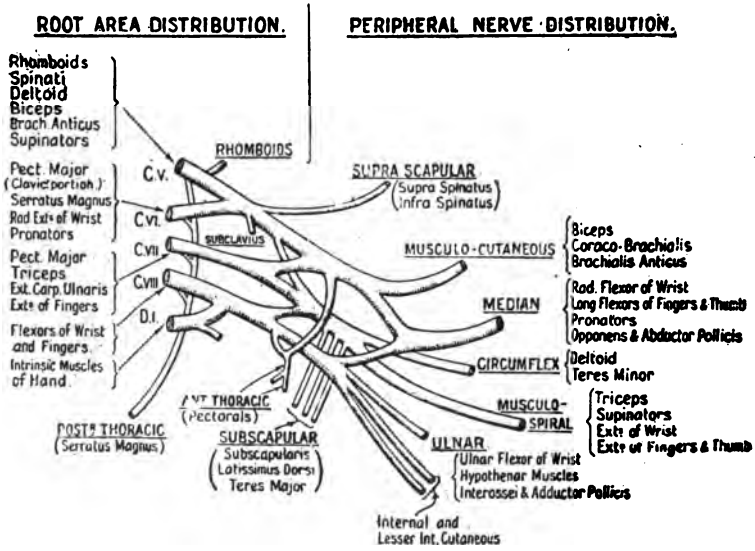


FIG. 185.—The motor supply of the brachial plexus, showing both its root-area distribution and its peripheral nerve distribution.

1. The *Erb-Duchenne syndrome* results essentially from a lesion of the anterior primary division of the fifth cervical nerve. Additional involvement of the sixth cervical seems to have a little effect on the extent of the paralysis. The muscles involved are those seen paralyzed in the so-called birth palsies; deltoid, biceps, brachialis anticus, and supinator longus, and in addition, some involvement of the rhomboids, spinati and sometimes of the pectorals. There is a more or less well-defined area of hypesthesia or anesthesia over the sensory distribution of the fifth and sixth cervical roots. (See Fig. 126 of sensory root areas.) Owing to the overlap of adjacent roots, however, this is variable in extent and is often wholly absent.

2. The *lower arm type* (*Klumpke*) results from a lesion of the eighth cervical and first dorsal roots or of their combined primary

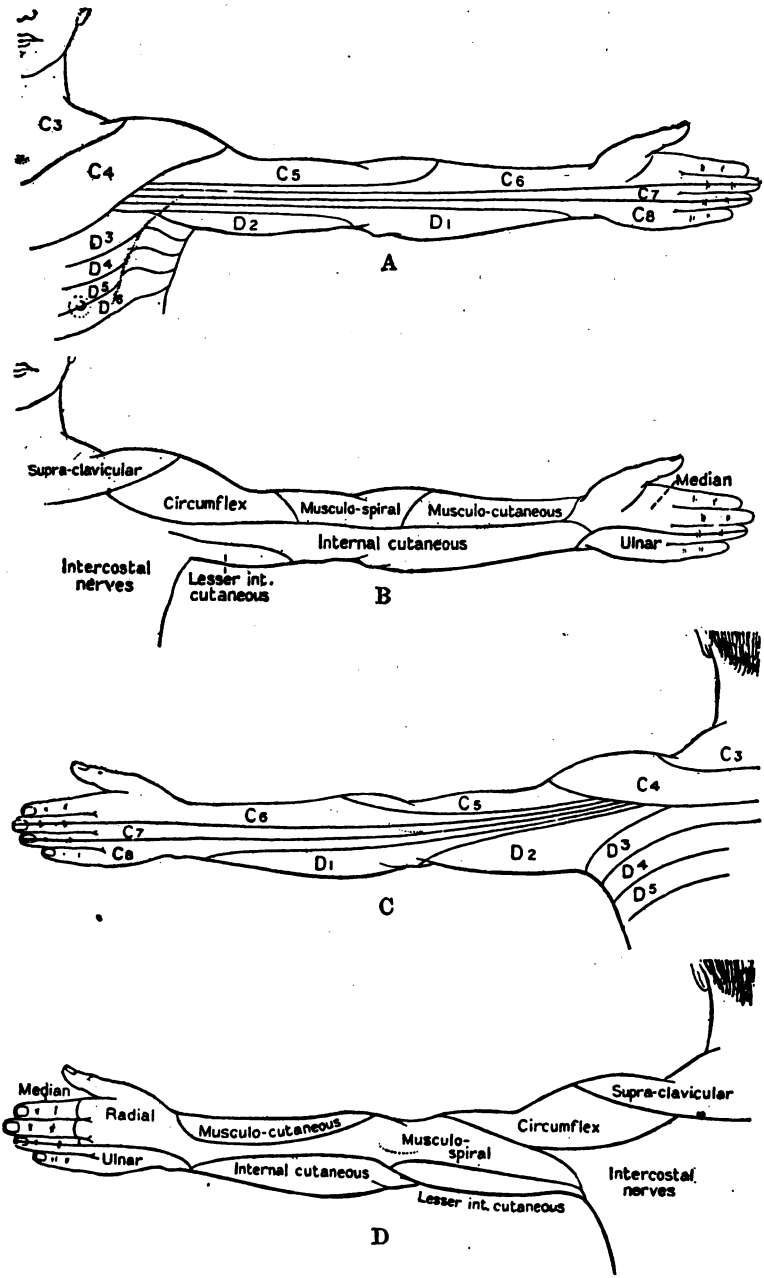


FIG. 186.—The cutaneous sensory supply of the upper limb. A and C show the areas supplied by the posterior roots; B and D the areas supplied by the peripheral nerves.

cord. Injury results in paralysis of all the small muscles of the hand (DI) with some involvement of the ulnar flexors of the wrist and fingers (CVIII). The sensory disturbances in the lower root lesions are much better defined than those in the upper root lesions and are confined to the inner aspect of the arm and forearm and hand, that is, in the root distribution of the eighth cervical and first thoracic nerves.

Oculo-pupillary symptoms accompany this lesion, as the sympathetic nerves for the superior cervical ganglion leave the cord with the first dorsal root. (See Cervical Sympathetic.)

B. *Lesions of the secondary cords.—Lesions of the inner cord* result in loss of sensation along the inner side of the arm, forearm and

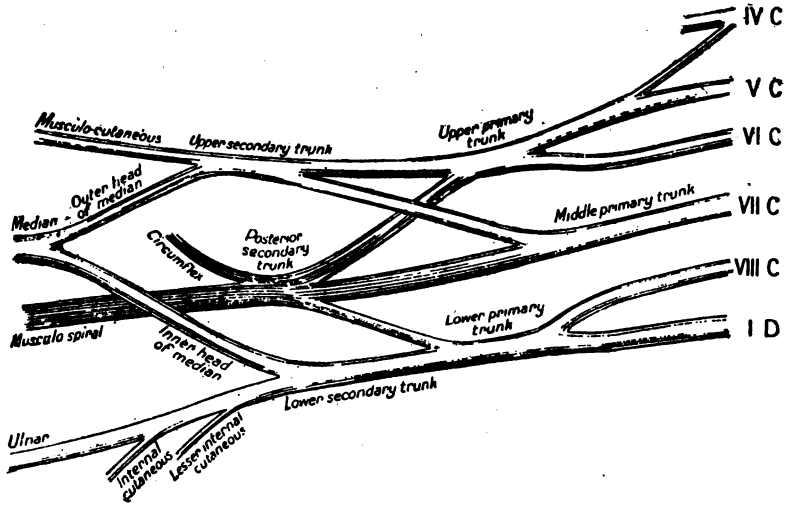


FIG. 187.—Constitution of brachial plexus.

hand as a result of involvement of the internal cutaneous and lesser internal cutaneous nerves. There is paralysis of all the intrinsic muscles of the hand and some of the flexors of the wrist and fingers, especially on the ulnar side. There is no involvement of the cervical sympathetic, as the white rami communicantes have already left the nerve trunks to join the sympathetic cord.

Lesions of the posterior cord result in paralysis of the muscles supplied by the circumflex and musculo-spiral nerves and loss of sensation in the region supplied by these nerves.

Lesions of the outer cord result in paralysis of muscles supplied by the musculo-cutaneous and the outer head of the median; biceps, coraco-brachialis, brachialis anticus, and flexors of the fingers and the wrist. There is anesthesia along the outer aspect of the forearm.

Cervical sympathetic.—The fibers of the cervical sympathetic leave the spinal cord by the eighth cervical and first dorsal roots. They

pass up the sympathetic cord to the superior cervical ganglion. In the neck they are behind the common carotid and internal carotid arteries.

Paralysis of the sympathetic results in the following: (1) The pupil is smaller than on the normal side. It does not dilate when shaded (paralysis of the dilator pupillæ); (2) there is drooping of the eyelid (paralysis of the smooth muscle fibers in the levator palpebral); (3) there is sinking in of the eyeball (paralysis of Müller's orbital muscle); (4) there may be reddening of the face, neck, and arm, and vasodilatation (paralysis of the vasoconstrictors).

Irritation of the cervical sympathetic produces: (1) Dilatation of the pupil; (2) widening of the lid slit; (3) exophthalmos.

Cervical plexus.—The cervical plexus is formed from the anterior primary divisions of the first four cervical nerves. From the plexus arise sensory nerves distributed to the skin of part of the head, neck, and shoulders (small occipital, great auricular, superficial cervical, and supraclavicular).

The chief muscular branch is the phrenic nerve. The phrenic nerve arises from the fourth cervical, descends on the scalenus anticus, enters the thorax and is distributed to the diaphragm.

Paralysis of the phrenic results in weakness of the movements of the same side of the diaphragm. The costal angle is usually more obtuse on the paralyzed side.

Sciatic nerve.—The sciatic nerve is the most frequently affected of the nerves in the lower limb. The bulk of its trunk and the length of its course renders it particularly liable to injuries. It arises from the fourth and fifth lumbar, first, second, and third sacral roots. The original branches are united at the level of the sciatic notch. It passes into the buttock covered by some of the deep muscles and the gluteus maximus. It descends along the back of the thigh to the popliteal space. At the upper end of the popliteal space it becomes superficial. At varying levels in this region, but often much higher, it divides into its terminal branches, the external popliteal and the internal popliteal nerves. In its course in the thigh there are given off, the nerve to the semitendinosus, which arises very high, and the nerve to the biceps and semi-membranosus, which generally arises somewhat lower. (Fig. 188.)

FIG. 188.

The external popliteal leaves the main trunk of the sciatic in the popliteal space and winds around to the outer surface of the leg. It

passes around the neck of the fibula to reach the antero-external surface of the leg, and at this level is very superficial. It divides into its terminal branches, the *anterior tibial* and the *musculo-cutaneous*, which pass down the antero-external aspect of the leg to

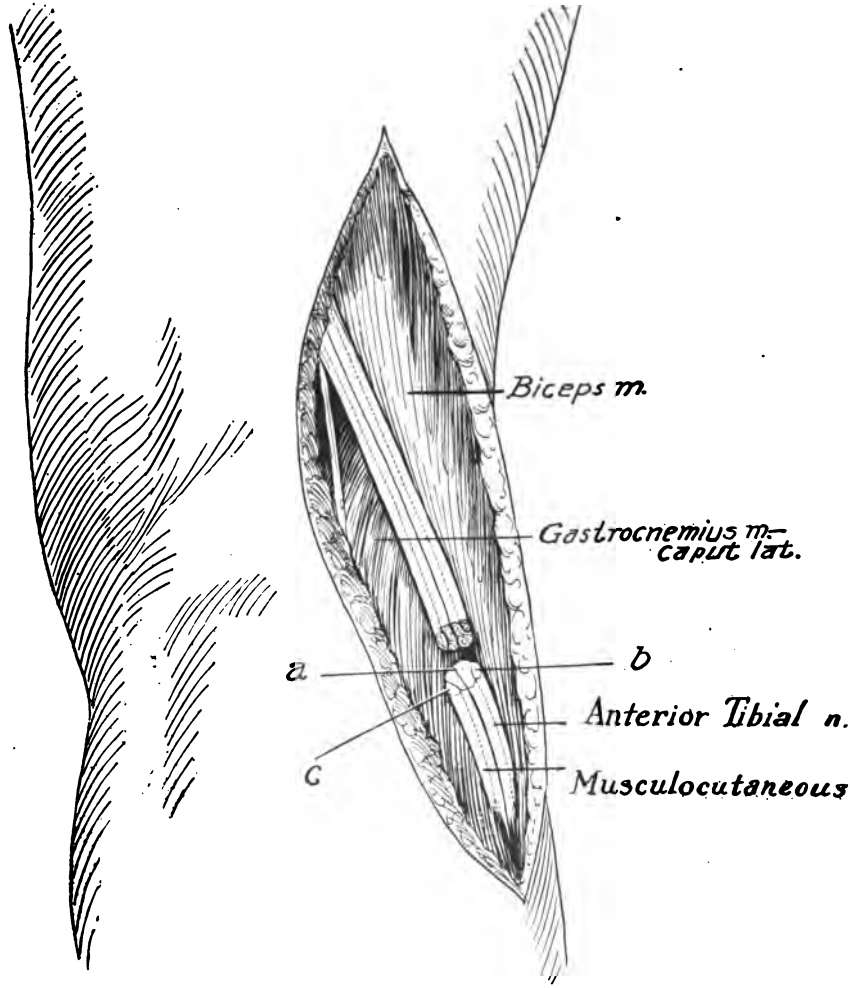


FIG. 189.—Location of nerve tracts in the external popliteal. Right popliteal space. The external popliteal divides into the musculo-cutaneous (medial) and the anterior tibial (lateral). In the former, the tract for the peroneal muscles have a dorsomedial position occupying about one-third of the cross section. In the cross section of the anterior tibial the tract for the anterior tibial muscle lies close to the biceps muscle. (Vulpius and Stoffel.)

the foot. The *anterior tibial nerve* as it descends along the front of the leg gives off branches to the tibialis anticus, the extensor longus digitorum and the extensor propius hallucis, and also to the extensor brevis digitorum. Its cutaneous branches anastomose with those of

the musculo-cutaneous, but it may have a small area of exclusive sensory supply occupying the adjacent surfaces of the dorsal aspect of the great toe and second toe.

The *musculo-cutaneous* supplies motor branches to the peroneus longus and peroneus brevis. Its cutaneous branches supply the antero-external surface of the leg and the greater part of the dorsum of the foot.

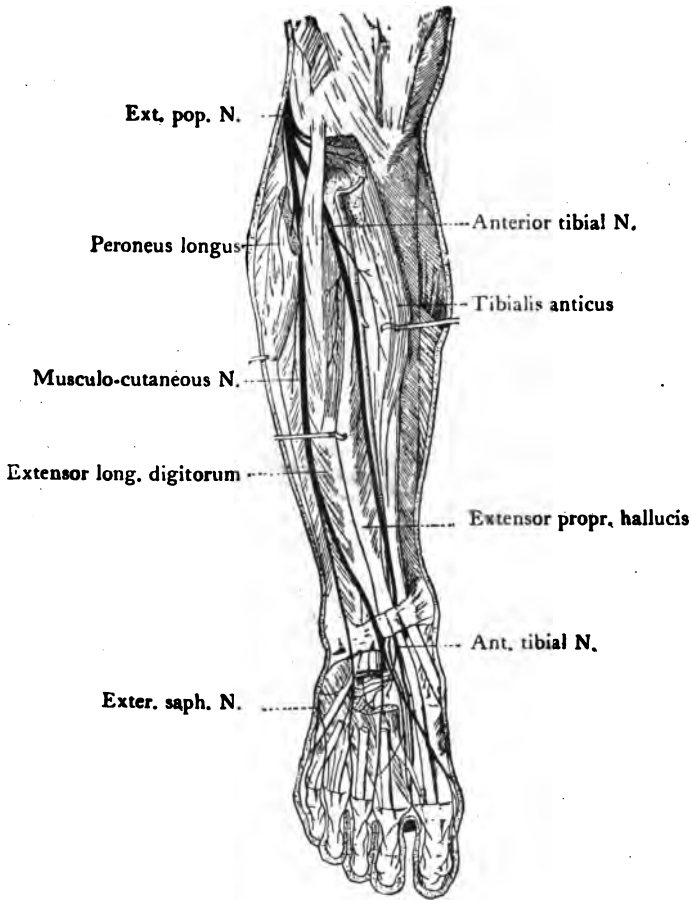


FIG. 190.—Musculo-cutaneous nerve and anterior tibial nerve. (After Hirschfeld, simplified.)

The *internal popliteal* descends through the popliteal space between the heads of the gastrocnemius, and passes beneath the soleus. It descends on the posterior surface of the leg between the superficial and deep layer of muscles. At the level of the ankle it turns inward, passes beneath the internal malleolus of the tibia and divides into its terminal branches, the internal and external plantar nerves.

The internal popliteal supplies the gastrocnemius, soleus, plantaris and at a lower level the tibialis posticus, flexor hallucis longus and flexor digitorum longus. In the popliteal space it gives off the *external saphenous*, which passes down superficially in the middle of the back of the leg and supplies cutaneous branches to the lower part of the back of the leg, lateral surface of the ankle and dorso-lateral surface of the foot. The internal and external plantar nerves

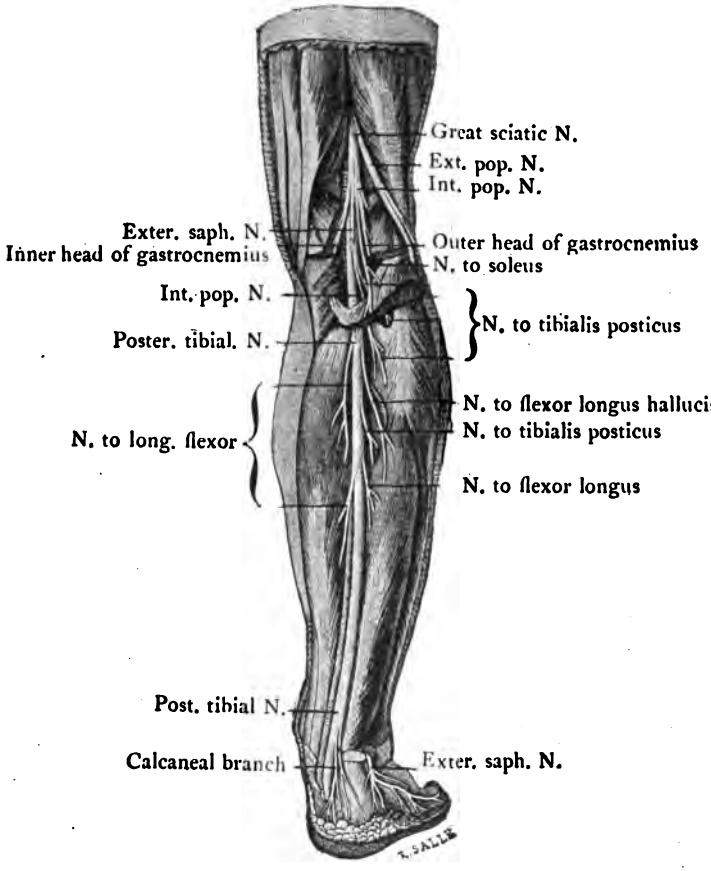


FIG. 131.—Internal popliteal nerve and posterior tibial nerve. (After Sappey.)

supply the muscles of the sole of the foot, sensation over the sole and the terminal phalanges of the dorsum of the toes.

External popliteal.—*Motor.*—Paralysis of the external popliteal results in loss of the power of dorsal flexion of the foot (tibialis anticus, extensor longus digitorum, and extensor hallucis longus).

Adduction of the foot, which is dependent on the combined action of tibialis anticus and tibialis posticus, is weakened because of the paralysis of the former. For the same reason the inner arch of the foot is weakened.

Abduction of the foot (elevation of the lateral border) is impossible, because of paralysis of the peronei.

The foot is in the position of talipes equino varus.

Sensory.—There is a loss of cutaneous sensibility over the antero-external aspect of the leg and dorsum of the foot. There is no loss of deep sensibility.

Dissociated syndromes may occur from partial lesions. Where the nerve lies behind the head of the fibula, the fibers for the tibialis anticus are in front, and for the peronei behind.

Anterior tibial.—The anterior tibial is the continuation of the external popliteal. It may be injured by wounds in the front of the leg.

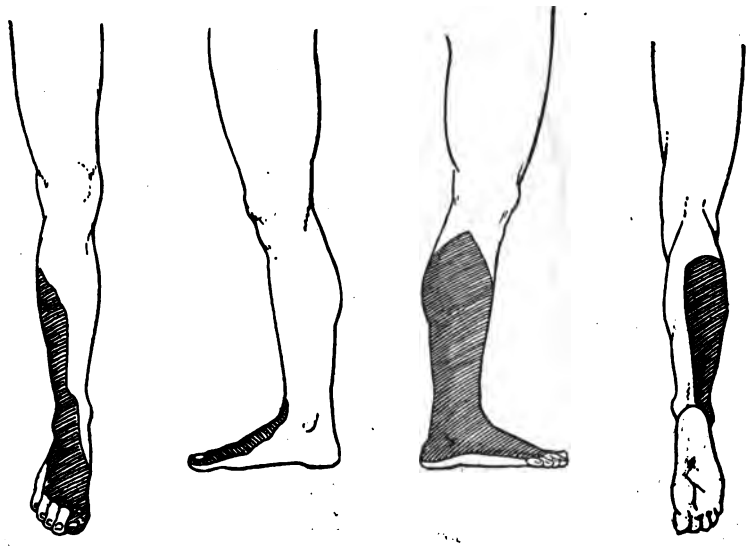


FIG. 192.—External popliteal paralysis. The shaded area shows the extent of anesthesia to pin-prick and cotton-wool. The nerve was injured by a piece of shell 1 inch below the sciatic notch. There was paralysis of the peronei, tibialis anticus, and extensors of the toes, with inability to dorsiflex the foot, evert the foot, or to extend the toes.

Complete division results in foot-drop. The foot is in the position of pes equinus. Sensory disturbance may be absent, or there may be hypesthesia over the inner side of the dorsum of the foot, especially the adjacent surfaces of the great and second toes.

Wounds low down in the leg may involve only the fibers for the extensor hallucis longus and extensor brevis digitorum.

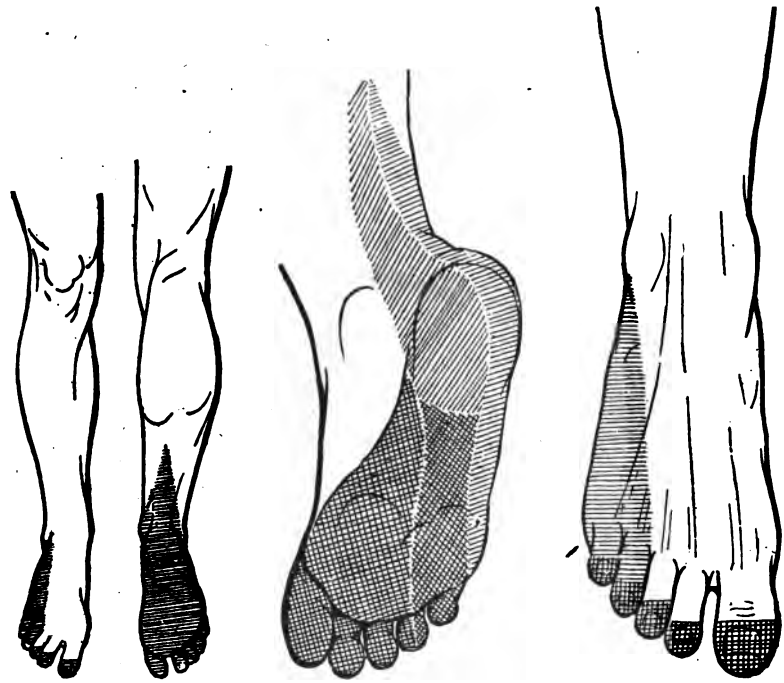
Musculo-cutaneous.—The nerve may be injured in wounds in the lateral aspect of the leg.

Motor symptoms.—Paralysis of the peronei. The foot is in the varus position. Abduction of the foot (raising the outer edge) is impossible.

Sensory symptoms.—Loss of cutaneous sensibility over the antero-lateral aspect of the leg and dorsum of the foot. The lateral aspect of the foot, supplied by the external saphenous, is spared.

Injury to the nerve below the point of origin of the nerves to the peronei will only give a sensory disturbance.

Internal popliteal.—*Motor.*—There is paralysis of the muscles of the calf and sole of the foot. There is loss of flexion of the ankle (gastrocnemius and soleus) and of flexion of the toes (flexor longus digitorum, flexor hallucis longus, and flexor brevis digitorum).



FIGS. 193, 194, 195, and 196.—Sensory area of the internal popliteal comprising: The external saphenous; external surface of the instep (horizontal hatching), outer edge of the foot, dorsal surface of the foot to the third intermetatarsal space. The posterior tibial, cutaneous branch (oblique hatching). The external and internal plantars (crossed hatching) which supply, on the dorsal surface, the last phalanx of the toes.

Adduction of the foot is weak (tibialis posticus) but not lost. The foot is in the position of talipes calcaneo valgus.

In walking there is no spring to the step, as the patient can not force his heel from the ground.

Sensory.—There is loss of cutaneous sensibility over the whole sole of the foot, back of the heel, and outer surface of the foot. There is no loss of deep sensibility. (Figs. 193, 194, 195, 196.)

Posterior tibial.—The posterior tibial is the continuation of the internal popliteal in the calf. In paralysis of this nerve the long flexors of the toes and tibialis posticus escape.

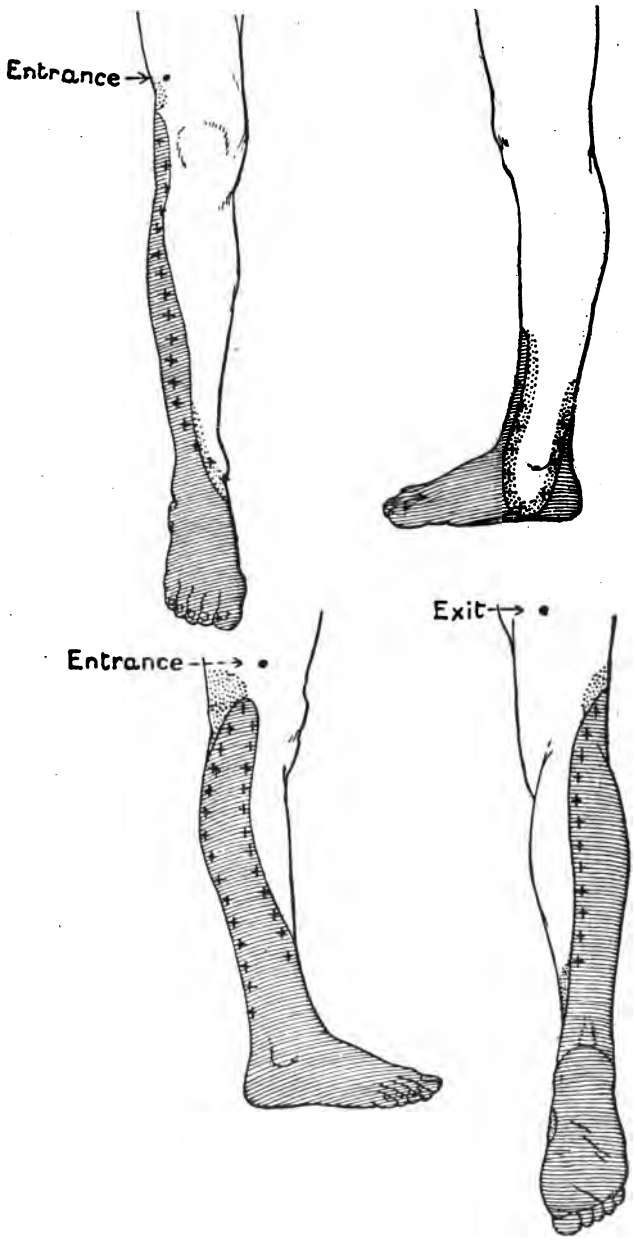


FIG. 197.—The sensory loss in paralysis of the great sciatic nerve. The shaded area is that insensitive to pin-prick, the dotted area is the additional area insensitive to cotton-wool; the crosses mark the loss of sensation to deep pressure.

Motor.—Only the muscles of the sole of the foot are involved. This may be readily overlooked unless electrical examination of the plantar muscles is made in all cases of wounds of the calf. There may be hyper-extension of the toes from overaction of the extensor longus digitorum.

Sensory.—Loss of cutaneous sensibility over the sole.

External saphenous.—This nerve may be affected alone, where it lies superficial on the back of the leg. Division of the nerve results

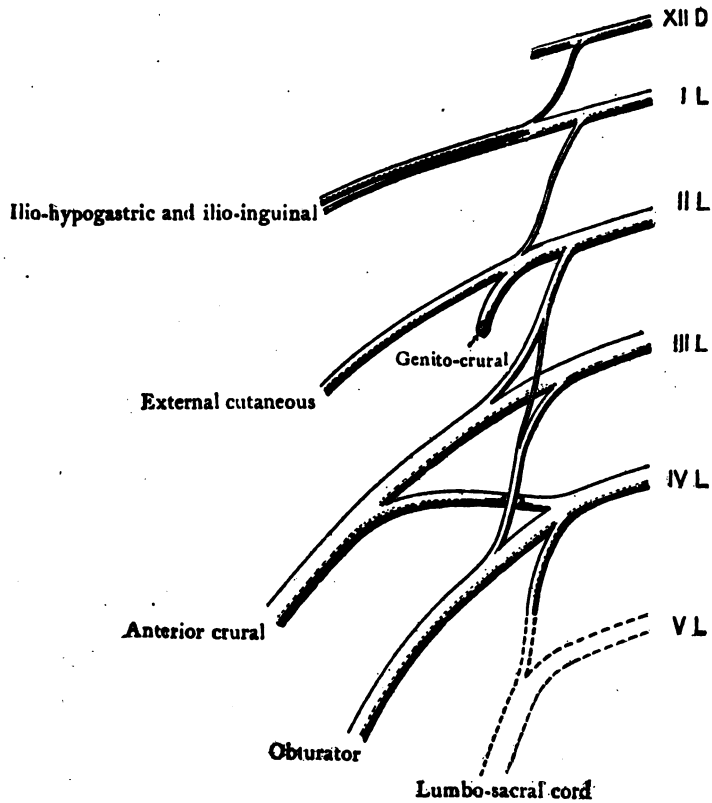


FIG. 198.—Lumbar plexus.

in loss of cutaneous sensation over the back of the ankle and lateral aspect of the foot, including a little of the dorsum of the foot.

Great sciatic.—Complete division of the sciatic will give a combination of the symptoms of division of the internal and external popliteal nerves. All movements of the foot and toes are lost. Division of the nerve in the thigh will result in paralysis of the hamstring muscles (flexors of the knee). The semitendinosus may escape, as its nerve is given off very high. Slight flexion of the knee is still possible through the gracilis (obturator) and perhaps also, the semitendinosus.

There is extensive loss of sensibility below the knee, the inner surface of the leg (internal saphenous) alone escaping.

Irritating lesions with marked vasomotor and trophic disturbances are common. Injuries of the sciatic trunk and of the median are the most frequent sources of causalgia.

Dissociated syndromes are frequent, owing to the size of the nerve. The external and internal popliteal, though bound together, are really separate nerves well up in the thigh. In injuries of the trunk the external popliteal is much more apt to suffer than the internal popliteal.

Lumbo-sacral plexus.—The lumbo-sacral plexus gives rise to all the

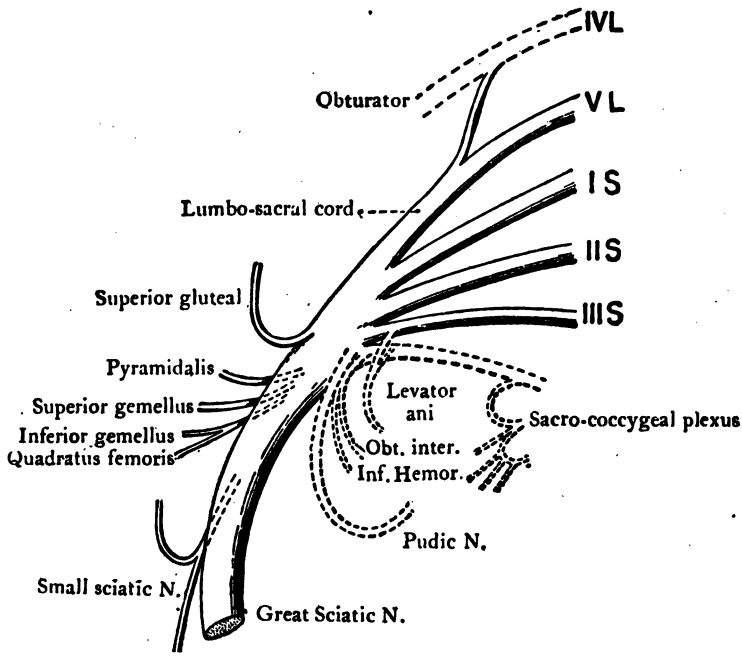


FIG. 199.—Sacral plexus.

nerves of the lower limb. It is composed of two parts, the lumbar plexus formed by the first, second, third, and fourth lumbar roots, (L. 1–2–3–4) and the sacral plexus formed by the fifth lumbar, first, second, and third sacral roots (L. 5, S. 1–2–3). There are anastomoses between all adjacent roots.

Lumbar plexus.—L. 1. Gives rise to the iliohypogastric and ilioinguinal.

L. 2. Receives a branch from L. 1 and gives rise to the external cutaneous and genito-crural.

L. 2, 3 and 4. Give origin to the anterior crural and obturator.

Sacral plexus.—This is made up of the lumbo-sacral cord (L. 4 and 5), and S. 1, 2 and 3.

The chief branch is the great sciatic nerve. Other branches are the superior gluteal from L. 4 and 5 and S. 1, which innervates the gluteus medius and minimus and the tensor fasciæ latæ; branches to the obturator internus, pyriformis, quadratus femoris and gemelli; the inferior gluteal nerve from L. 5, S. 1 and 2, which supplies the gluteus maximus. The roots which go to form the plexus may be affected either in the pelvis or in the cauda equina. Injuries to the roots will produce symptoms differing from those of the peripheral nerves because each of these roots supplies parts of several nerves and each nerve receives parts of several roots. The sensory disturbance is of the root or segmental type and not of the peripheral type.

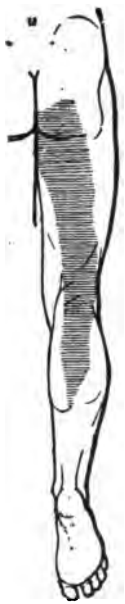


FIG. 200.—Sensory distribution of the small sciatic nerve.

Injuries to the upper lumbar roots, L. 1 and 2, result in weakening of the iliopsoas (flexion of the thigh).

Injuries of L. 3 and 4 result in paralysis of extension of the knee and adduction of the thigh through the anterior cural and the obturator.

Injuries of L. 4 and 5 will involve the glutei (adduction of the leg), tensor fasciæ latæ and some of the leg muscles.

According to Sherren and Stewart, the tibialis anticus receives its chief supply from L. 4, and the other muscles of the antero-external surface of the leg (extensor longus digitorum, peronei) from L. 5. Certainly the tibialis anticus receives its chief supply higher than the others. Thus, in lesions of L. 3 and 4 the tibialis anticus may be the only leg muscle to suffer, and in lesions of L. 5 and S. 1 the tibialis anticus may be the only leg muscle to escape.

S. 1 and 2 supply the muscles of the sole and calf and the hamstrings.

S. 3 and 4 supply the bladder and rectum.

Hematomyelia in the lumbo-sacral region is not infrequent as the result of a severe blow over the lower thoracic or upper lumbar spine. It may produce symptoms resembling lesions of the plexus or cauda. As the hemorrhage usually involves the anterior horns it will produce a paralysis of the lower motor neuron type. By involving, as it infrequently does, the posterior horns it will produce a sensory disturbance of segmental type. However, this sensory disturbance may be dissociated (loss of pain and temperature with preservation of touch), in which case it will be easy to differentiate from root lesions. If the site of the injury is below the second lumbar spine hematomyelia is unlikely, as the injury is below the level of the cord, and root or cauda lesions are most probable.

Small sciatic.—The small sciatic arises from the first, second, and third sacral roots. It leaves the pelvis with the great sciatic and descends in the back of the thigh to the upper calf. It is purely a sensory nerve. Division of the small sciatic causes a loss of cutaneous sensibility over the back of the thigh, buttock and perineum.

Anterior crural.—The anterior crural nerve arises from the lumbar plexus. It crosses the iliac fossa, and enters the thigh beneath Poupart's ligament, lateral to the great vessels. Under Poupart's ligament it divides into its several branches to the muscles and skin of

FIG. 201.

FIG. 202.

FIG. 203.

FIGS. 201 and 202.—Sensory region of the anterior crural. Above the knee, region of the anterior crural proper. Below the knee, region of the internal saphenous.
FIG. 203.—Sensory disturbance in complete interruption of the internal saphenous nerve in Scarpa's triangle.

the thigh. One branch, the internal saphenous, continues on down the inner side of the leg and foot.

Motor.—Quadriceps extensor, sartorius, pectineus and branches to the psoas and the adductors.

Sensory.—Several branches supply the antero-internal surface of the thigh.

The internal saphenous nerve continues on down, crosses the inner side of the knee, and descends on the inner aspect of the leg to the foot.

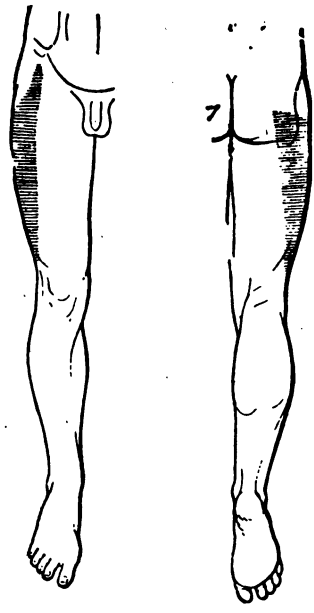
Injury to the nerve is comparatively rare. Complete division can only occur in the pelvis. In the upper thigh it has already

broken up into its several branches, and an injury will affect only a few of these.

Complete division.—The chief symptom is loss of extension of the leg (quadriceps extensor). The patellar reflex is lost.

Sensory.—Loss or impairment of cutaneous sensibility over the antero-internal aspect of the thigh. Loss of cutaneous sensibility over the inner aspect of the knee, leg and foot.

The internal saphenous nerve may be injured alone in the thigh or leg. Division will result in loss of cutaneous sensibility over the inner aspect of knee, leg and foot.



FIGS. 204 and 205.—Sensory region of the external cutaneous.

Obturator nerve.—The obturator arises from the lumbar plexus. It crosses the brim of the pelvis, and enters the thigh by the obturator foramen.

Motor: Adductors of the thigh and the gracilis.

Sensory: A small area on the lower inner aspect of the thigh.

Complete division of the nerve results in loss of power in adduction of the thigh. The loss is not complete, as some fibers to the adductors arise from the anterior crural and sciatic. There is generally no loss of sensation, owing to overlapping with adjacent cutaneous nerves.

External cutaneous nerve.—The external cutaneous arises from the lumbar plexus. It crosses the pelvis high up and enters the thigh below Poupart's ligament. It is purely sensory and supplies the upper outer portion of the thigh. Section of the nerve results in loss of cutaneous sensibility in this region. Neuralgia of the external cutaneous (meralgia paresthetica), is not an uncommon affection. It is characterized by spontaneous pains in the region supplied by the nerve.

Genito-crural.—The genito-crural nerve supplies sensory fibers to the middle of the upper part of the thigh and the scrotum. Division of the nerve results in no loss of sensation, but may cause pains in the upper thigh and in the scrotum.

Iliohypogastric and ilioinguinal nerves.—The iliohypogastric and ilioinguinal nerves arise from the first lumbar root. They are sensory nerves, but with no area of exclusive sensory supply. Injury to the nerves results in no loss of sensibility, but may give rise to pains in the upper outer and upper inner portions of the thigh.

The after-care of nerve injuries.—Amid the abundant literature of nerve surgery which the war has produced one is struck by the meager attention given to after-care. There are two obvious reasons for this. First, the time is still too short for the systematic study of end results. Second, an over-enthusiasm for operating has diverted surgical interest from the more prosaic but no less important subject of convalescence. With the shifting personnel of military hospitals, too often an operator never has the chance to follow through a sufficient proportion of his cases to develop an intelligent and systematic scheme of after-care.

The technic of operations on wounded nerves is undeniably one requiring a high grade of surgical skill, but the contribution toward functional restoration made at the time of operation may be completely nullified by lack of subsequent care. For example, in the case of a musculo-spiral paralysis, if the wrist drop is allowed to continue during the many months of nerve regeneration, the extensor muscles will undergo fibrosis and, though the nerve may show complete recovery, there is no muscle fiber remaining to contract, the operation is worthless, and the functional result is a complete failure.

It is well to fix in the mind at the outset of any study of nerve surgery that it differs radically from the brilliant results of most operative work, which when successful yields to patient and friends almost immediate evidence of improvement. It is a painful and discouraging thing to undergo a tedious and exhausting operation with the knowledge that even if a complete success it will show absolutely no results from 6 to 12 months; and the suspense of waiting those many months for the decision as to whether the effort is to prove a success at all is a gruelling test of fortitude for patient and surgeon alike.

Too much stress can not be laid on the importance of a thorough understanding of all the measures at the surgeon's disposal for the after-care of these critical cases. An appreciation of the relative value of these various methods is very necessary for their successful use. Proper sequence in their employment and the amount of time to be devoted to each should be carefully understood, for as much harm may be done by their over-use as by their neglect.

The after-care of nerve injuries involves two problems: First, the care of freshly wounded and post-operative cases; second, the care of late neglected cases where muscular contraction has taken place and deformity has already resulted. In one case treatment is preventive, in the other corrective. Many neglected cases may require secondary operative procedures.

Type of injury. Nerves may be completely or partially cut or torn across; so seriously bruised that function is interrupted temporarily

or permanently; strangulated by pressure from contracted scar tissue. Greater or less degrees of paralysis are present according to the type of injury. The ends of several nerves may be separated by considerable distances, and operation is often a necessary preliminary. Post-operative treatment does not differ from that required in cases which do not call for operation, and both classes may be grouped in considering after-care.

Upon the interruption of nerve function, the following effects on muscles are observed: (*a*) Tone and contractility are lost; (*b*) atrophy sets in due to (1) lack of voluntary action, (2) alteration in nutrition; (*c*) deformity due to (1) stretching of the paralyzed muscle, (2) contraction of healthy muscle.

(*a*) *Muscle tone* is a condition of normal tension of muscle fiber due to the integrity of the reflex arc. When the nerve is completely cut the muscle tone disappears and the elasticity of the muscle group is lost. By the preservation of the normal tone of opposing muscles which maintains them in a state of slight contraction, paralyzed muscles become stretched and very soon lose the power to regain this normal tone. Hence the great need to prevent the overstretching due to faulty posture during convalescence.

Contractility is the power inherent in normal muscular tissue of contracting under stimulus. Direct mechanical stimulus will arouse this contractile power, but nerve energization is the normal stimulus. The integrity of the muscular protoplasm depends on exercise. Paralyzed muscle fiber rapidly loses its normal chemical and physical characteristics unless it is kept exercised by mechanical or electrical means.

(*b*) *Atrophy* from disuse does not differ from the wasting seen in any prolonged period of inactivity. There is in addition an atrophy due directly to nutritive changes from loss of innervation. This affects muscle, bone, and skin and is apparent in the glossy and often cyanotic appearance of the latter.

(*c*) *Deformity* occurs first as a result of flaccidity of paralyzed muscle groups and may be passively corrected. The habitual position is that produced by strong or extreme contraction of the unparalyzed groups. If this condition is allowed to continue without treatment, contractures occur in the active muscles which prevent correction of the deformity. At the same time, the paralyzed muscles lose their tone and elasticity. If proper treatment is neglected during convalescence, the nerve may recover but leave a functionally useless limb because of the development of a permanent deformity which interferes with effective muscular activity.

It is very necessary to emphasize the fact that degeneration of muscle begins immediately after injury. Treatment, therefore,

should be inaugurated at once and should be continuous. The objects to be achieved are: A. In recent cases—(1) prevention of deformity; (2) restoration of function. B. In old, untreated cases—(1) correction of deformity; (2) development of any remaining function.

At the outset, the fact must be faced that treatment is nearly always prolonged and often discouraging. The repair of sutured nerves requires, roughly, a year or longer, and the surgeon's hardest task is to maintain his own and his patient's enthusiasm during the long convalescence. Continuity of treatment is of the utmost importance. Relaxation for a week may undo many weeks' constructive work.

There is comparatively little to do for the repaired nerve other than to prevent immediate postoperative injury due to rough handling, and to favor the growth of the nerve tendrils by measures improving general health and local circulation. If a nerve has been resected and so shortened that suture is possible only after relaxation by flexion of the limb, great care should be observed not to tear the suture line by failure to maintain proper posture for a sufficient time after the operation. For two weeks or more motion may be inadvisable. Later it should be attempted gradually, and carefully controlled. Fortunately a nerve appears to be capable of very considerable stretching without harm if the process is carried on gently and slowly enough.

Nerves regenerate through rapid proliferation of the cut neurons from their proximal ends. The growth is through light granulation tissue easily injured, and the need of most careful treatment about the seat of active proliferation must never be forgotten. Even after fairly active treatment of neighboring structures has begun it is well to allow several weeks of consolidation before beginning local treatment about the seat of suture. Hemorrhages in the new formed tissue may lead to injurious cicatrization. It is for the same reason that an absolutely dry field is advised before suture of the skin wound after operating on a traumatized nerve.

When the early stage of repair is past, and growth of the nerve is well under way, further treatment directed specifically to the nerve itself is unnecessary. Then begins the *important* period of after care. This concerns itself with the treatment of *muscle*, *tendons*, and *joints*.

It may not be out of place to lay emphasis on the very great importance of pre-operative mechanical treatment of such cases as have through neglect developed deformity. No greater surgical mistake can be made than to operate and expect satisfactory results on divided nerves where muscles have been allowed to contract and become fibrous, where scars bind down important tendons, where joints are

ankylosed, unless these conditions are first relieved. There is scant use for the innervating mechanism if the machine itself is wrecked. Not rarely this preliminary work will require many weeks and possibly some operative work on other structures before the field is satisfactory for undertaking suture or dissection of the nerve.

Methods of treatment.—The methods of treatment employed are protective and corrective splinting, and the various forms of physiotherapy.

Preventive splinting.—Rest in splints which hold the part in an over-corrected position is essential to prevent deformity in recently paralyzed muscles. The position as given by Sir Robert Jones and perhaps most generally accepted is one of maximum over-correction with the paralyzed muscles completely relaxed and the active muscles held fully stretched to overcome their tendency toward contraction. Sir Harold Styles, however, makes the following statement:

"Adhesions too often follow the application of splints to produce relaxation of the paralyzed muscles. In these cases, hyper-relaxation has been produced. Had only complete relaxation been produced, the opposite structures would not have been stretched and no adhesions would have developed.

"Physiological rest is the only position in which one or more groups of paralyzed muscles can be completely relaxed without stretching the opposing structures. In this position the hand should be placed in all lesions of the musculo-spiral, median or ulnar. No adhesions form, and recovery in the paralyzed muscles is as rapid as when they are hyper-relaxed."

Types of splints.—The standard splints supplied by the Army Medical Service will be found sufficient for most requirements. Adaptations of these will suggest themselves to the surgeon to meet particular needs. These splints have been adopted after much critical study, and embody the results of three years of splint evolution in the British and French service. It must be distinctly understood that they do not represent the only good splints, but merely one simple and efficient set suited to most of the purposes for which splinting is necessary. In addition to these, plaster of Paris is a most useful splinting material and one of great value in meeting the need for unusual postures often demanded in the after-care of nerve injuries. Light weight material is very important in providing splints, as they must be worn over long periods and many of the cases are, of course, ambulatory, but this principle must not be carried so far that durability is in any way sacrificed. Ease and speed in application are most desirable, since splints must be removed frequently for massage and other treatment.

Much ingenuity has been expended in the invention of adjustable splints fitted with hinges, turn-buckles, springs or other recognized

mechanical devices for altering position or tension. In general these types will prove disappointing. The spring principle is rarely of great value and is always attended with some risk. Adjustable mechanisms are prone to disabilities of their own and in a busy ward are difficult to keep in constant repair. Of far more general service is the rigid splint which may be bent to a set contour and altered from time to time by further bending or by the gradual addition of felt padding as alteration of posture is secured. It is very desirable to arrange an ambulatory apparatus for all cases so far as possible on account of the long period necessary for convalescence.

Application of splints.—Each case will present its individual problem and calls for special modification in the use of splints, but the following principles of treatment may be of service in approaching the more frequent types of paralysis:

Circumflex nerve.—Injury to the circumflex nerve results in deltoid paralysis. The shoulder sags under the weight of the arm and power of abduction is lost. The arm should be maintained in a position of abduction at right angles to the body. To accomplish this, a splint molded to the trunk is necessary, and it should take its bearing from the crest of the ilium on the affected side, otherwise it will slide downward when the patient is in the upright position, and lose its efficiency. From the body portion of the splint a right-angled armpiece runs outward at the axilla in a plane a little anterior to the coronal plane of the body and on this rests the arm. The splint supplied for the Army—Jones adduction arm splint—is well adapted to the purpose, though plaster with wire armpiece is perhaps more commonly employed and is both comfortable and efficient.

Musculo-cutaneous nerve.—Paralysis of the anterior muscles of the upper arm prevents flexion at the elbow and results in stretching of the biceps. This is treated by retaining the elbow at right angles by means of a Jones right-angle elbow splint with hand piece for support of the hand in flexion, extension, supination, or pronation, as desired. It is, of course, necessary to provide ambulatory cases with a sling to give comfortable support to the apparatus.

Median nerve; ulnar nerve.—These nerves supply the flexor muscles of the forearm. When paralyzed the tendency will be toward hyperextension of the hand on the wrist and the fingers on the hand. To offset this a splint should be worn to maintain the hand and fingers in flexion. A reversed short cock-up splint with a ball or a roller bandage held in the palm of the hand will serve. A plaster splint is more convenient and is easily removed and replaced when necessary. Moderate pronation should also be provided, on account of the loss of the pronator teres.

Musculo-spiral nerve.—Loss of the musculo-spiral nerve results in the paralysis of the extensors of the arm, forearm and hand. The

typical signs are wrist-drop and partial loss of supination. Hyperextension of the wrist is necessary. In high injury of the nerve the triceps will suffer. Gravity is usually sufficient treatment for this latter. To secure hyperextension at the wrist a cock-up palmar splint is necessary. Various types are available, but the short cock-up of Jones is best adapted for the purpose. This splint has a short arm-piece and a palmar portion bent to an angle of from 45 to 70 degrees. The palmar piece should be convex backward to fit and support the palmar arch and made narrow enough to avoid impinging on the thenar and hypothenar eminences and so allow maintaining of the palmar arch and allowing support and adduction of the thumb. It should be short enough to avoid interference with metacarpal joints.

Anterior crural nerve.—Paralysis of the anterior crural gives loss of power in the anterior muscles of the thigh and the iliacus and pectineus which act as thigh flexors. Kicking power is absent, and contraction of the flexors of the knee is to be expected. If a splint is needed, the leg should be held extended on the thigh by means of a straight posterior splint of wood or plaster of Paris or by a Thomas knee splint, which is light, easy to apply, and comfortable to wear.

Sciatic nerve.—The sciatic supplies the muscles of the back of the thigh. Paralysis allows hyperextension of the knee, best guarded against by a Thomas splint. At the same time there is complete paralysis of the muscles moving the foot, and retention at right angles is necessary by means of a metal or plaster foot piece.

The internal popliteal nerve.—As the tibial and posterior tibial nerves this supplies the plantar flexors of the foot. Its paralysis results in dorsi-flexion or calcaneus. To overcorrect this position the foot must be held in strong plantar flexion. This is accomplished by means of a Jones combined ankle and lower leg splint with foot piece properly extended. The short ankle splint gives sufficient fixation for many cases and is rather lighter and more comfortable to wear.

The external popliteal nerve.—Paralysis of the anterior tibial branch, supplying the dorsal flexors, allows foot-drop and eversion. This may be met by hyperextension and inversion, which may be accomplished by bending upward the foot piece of the Jones short or long leg splint, or is frequently accomplished by means of plaster. Musculo-cutaneous nerve paralysis produces inversion through loss of the peroneal muscles and a position of the foot approximating clubfoot. Overcorrecting clubfoot braces are useful, though here again the ankle splint may be adopted.

The splints should be comfortable and worn constantly, save when treatment is being given. During the late stages of convalescence they should be gradually removed, at first for short periods during

the day, when the patient is allowed active exercise. They should be worn at night for a prolonged period after recovery is well advanced. This extends into many weeks or months, and it is very important to keep the patient under observation during this period of splint removal, as contractures may occur long after recovery of muscle power and tone is apparently complete.

Corrective splinting.—In an old deformity resulting from paralysis of nerve there are three tissues entering into the contracture—ligament, tendon, and muscle. Ligaments are nonelastic; muscle possesses elasticity. Care must be taken that nonelastic tissue is not unnecessarily torn by efforts at forcible correction. On account of nutritional changes, affected structures atrophy and become less resistent to trauma. There is danger of rupture of ligaments and even avulsion of bone at the point of ligamentous attachment. For this reason, non-operative correction of deformity is always preferable in cases where it can be accomplished by this method. It is a slow and often tedious process, but eliminates the danger of injury to atrophic muscle, ligament, and bone, which may occur from rough handling under ether. Many patients have been through several operations and would themselves prefer a slower convalescence to further operative interference.

If proper patience is exercised, successful correction of very extensive deformity may be accomplished by gradual stretching of contractures with splints. The initial selection and fitting of the splint is of great importance, and accepted principles of splint application must be kept constantly in mind. Corrective pressure is borne on soft tissues, and the amount that can be applied is limited. Constant watchfulness is required to avoid skin or tissue necrosis. The splint should be removed at frequent intervals and regular physical treatment given both for the purpose of forestalling pressure injury and to increase pliability and local circulation. Skin sensation may be absent or lessened, and many patients are quite uncomplaining. The vigilance of the attending surgeon should be constant to assure as rapid correction as the skin resistance will permit. Adjustments left to the control of the patient are unwise, and the judgment of nurses and orderlies must not be trusted too implicitly. It is of great importance to gain the cooperation of the patient himself by explaining the purpose of the splint and the necessity for continuous treatment; otherwise he may loosen splints at night and retard his own recovery.

Physiotherapy.—Considerable space has been given to the problem of splinting, because in the early after-care as well as throughout treatment it is a consideration of the first importance. It is, however, but one phase of treatment, and the restoration of function

depends on a number of other factors which may be grouped under the general term, physiotherapy. Fortunately there is a considerable range of these methods, all of much value, and their variety is most desirable when one takes into account the length of treatment required in nerve cases and the problem of keeping the patient contented during that tedious period. The health of our motor organs depends on use. Degeneration follows disuse whether from paralysis or simple laziness. The repair of diseased organs is a nutritional problem. Hence, exercise and hyperemia are the two principles underlying all physiotherapeutic treatment. There are other elements that may make the treatment more effective, as, for example, a mild suggestion to the patient regarding the mysterious powers of electricity when we wish to maintain his interest over a long period of electrotherapy. The physicion should beware of the easy self-deception that enthusiasm for a special method produces, and should attempt a fair valuation of all recognized methods and train himself in a rational use of the different varieties.

Thermotherapy.—Heat is a recognized therapeutic agent for the production of hyperemia. The particular form of heat probably has less significance than its duration and the temperature reached. Hot water, hot air, and electric heat are all useful, the latter of widest application from its convenience. Actual temperatures in the heat chamber may be very high, the evaporation immediately about the skin preventing burning. The small portable electric-light heaters may be applied conveniently to any part of a limb. Various colored lamps have been advocated as possessing special virtues, a claim that has received little support. In the treatment of nerve cases the chief value of heat thermotherapy is by way of preparation for massage.

Hydrotherapy.—The use of baths and general hydrotherapeutic measures has attained marked prominence in the English general hospitals. In addition to the usual tub, sitz, and shower baths there are hose sprays, whirling baths with and without aeration, and other variations of the general principle of hydrotherapy. The object is stimulation of circulation by means of heat and cold contrasts and the mechanical effect of moving water on the skin. There is no doubt of the invigorating effect of hydrotherapeutic measures judiciously employed, and here again an important value is their use as a preliminary to massage.

Massage.—This is the most useful and generally applicable method of physical treatment in these cases. Many inventions have sought to supersede the human hand as instruments of massage, but without success. Attention should first be paid to maintaining the nutrition of paralyzed muscles. The overlying skin, often atrophic, benefits equally from the treatment. As explained in discussing the pathology of nerve repair, great gentleness should guide the masseur

during the early stages of work with recent injuries. Effleurage and wholly passive treatment with the part retained by a proper splint is followed gradually by more vigorous methods during temporary removal of the splint. Work should be done with the paralyzed muscle relaxed and the deformity overcorrected, and the patient's position while being massaged must be wisely planned. For example, in wrist-drop have the hand in supination, and the reverse in paralysis of the median and ulnar nerves. In circumflex involvement, the patient should be lying on his back and the arm raised beyond a right angle in abduction. Likewise, in foot-drop a prone position with foot hanging over the edge of the table will lessen the tendency toward plantar flexion, or the patient may be made to support the foot at right angles by holding the ends of a bandage looped about the toes.

The selection of efficient operators is a matter for careful consideration. Not only should we insist on well trained and experienced masseuses, but they should not be expected to undertake too many cases in a day. It has been found in English hospitals that one operator can care for about fifteen cases. If more than twenty cases are assigned to one masseuse the quality of her work deteriorates noticeably.

Corrective exercises should follow the gentler forms of massage necessary in freshly wounded cases, and are begun at once in older cases with deformity already developed. Force should be wisely graded, as it is in this type of work that much damage may be done through the use of too vigorous methods. Resistive and assistive exercises are of great benefit in the development of muscular strength and the reëducation of the patient in the use and control of muscle.

Finally, it is the duty of the masseuse to follow up the patient even after he has recovered sufficiently to begin active exercise and training in the curative workshop. Supplementary massage and direction is often of great assistance at this time in hastening the wounded man along the road toward the complete restoration of voluntary function.

Electrotherapeutics.—During the present war the use of electricity has received much attention and has achieved excellent results in the treatment of the after effects of injuries to the peripheral nerves, of injuries of the joints, and of functional nervous conditions. Many of the methods of application advised require complicated apparatus and a considerable degree of skill and experience in those who administer them. In the following pages only the more commonly used methods and those whose value has been proven will be discussed. The following forms of current are used: The constant or galvanic, the induced or faradic, the sinusoidal, the static and high frequency. Of these the constant is probably the more generally useful.

The constant current.—This is one which flows constantly from the positive anode to the negative pole or kathode, so long as the circuit is closed. It, therefore, always passes in one direction. The source of supply may be either a battery composed of 20 or more dry cells or, what is preferable when possible, an electric light circuit. If this is alternating it must be rendered unidirectional by a suitable converter. A current controller is preferable, although not essential. There must, however, be a means of gradually increasing and decreasing the strength of the current. If possible, a milliampere meter should be in the circuit. A switch by which the circuit can be opened or closed and the polarity reversed is essential. With this current each pole has certain physiological actions which it is well to remember.

The positive pole exercises a sedative influence over muscle and nerve (anelectrotonus).

Either pole will cause muscular contraction, but to do so the circuit must be either suddenly opened or closed. If the current passes without interruption or is gradually increased or decreased in strength no contraction occurs.

Faradic current.—This is characterized by more or less rapid alternations and a momentary duration of the current, it being manifest only during the instant of opening and closure of the primary circuit. When possible it is well to have an apparatus that allows both slow and rapid interruptions. This current has no polar influence as has the galvanic, and its principal use is as a means of exercising weakened muscles. It must be remembered that it will not, as a rule, cause contraction of muscles that are paralyzed, owing to a lesion of the peripheral neuron.

If the rapid interrupted current is employed more or less tonic or tetanic contractions is caused. The current slowly interrupted gives clonic contractions.

A type of coil which furnishes a current sufficiently weak to avoid painful sensations and the resulting undesired reflex contractions of adjacent muscles and which also allows the accurate regulations of current strength has been developed by Capt. Bristow, of the Royal Army Medical Corps, and is in extensive use in Great Britain. This obviates the chief objection to the faradic current and makes the faradic and galvanic sinusoidal currents unnecessary.

All who have written concerning injuries to the peripheral nerves occurring during the present war emphasize the great importance of this means of maintaining and increasing the nutrition of the affected muscles as well as exercising them and preventing adhesions in tendon sheaths and joints. In the case of divided nerves it should be employed in the interval before operation (when secondary suture is performed) as well as after it. In these cases ionic medication may

also be useful. It is applied over the seat of injury. In spinal-cord injury, if the cord is not completely divided, exercise of the paralyzed muscles may sometimes be of service. In cerebral injury with resulting hemiplegia it is useful, and then as a rule should only be applied to the extensor muscles, as by so doing contractures may be prevented or lessened.

High-frequency currents.—These are produced by discharging one or more condensers through a coil or wire. The condensers are charged either by an induction coil, static machine, or from an electric-light circuit, the current of which is stopped up by a transformer. The current is alternating or oscillating, and the frequency of these is very great. An induction coil may give a current interrupted several hundred times a second; the interruptions of these may amount to millions a second.

One of the peculiarities of these currents is that the human organism is not injured by them when a current strength of one or more amperes is used. With other forms of current this might be fatal. Their practical value in surgical cases is their power to produce local hyperemia and heat. They can not be entirely insulated by ordinary means, as, for instance, glass. If used properly they are not painful. According to the method by which they are obtained they are known as the D'Arsonval, Usinator and Tesla currents.

The heat obtained from the D'Arsonval apparatus differs from that obtained by other means of heating the body in that it penetrates, the degree of heat being greater within than it is on the surface and being dissipated very slowly. Therefore all the tissues between the two electrodes are heated, especially those of the deep structures. This is known as *diathermia or thermopenetration.*

The indications for the employment of these currents, especially diathermy, are neuritis, arthritis, synovitis, myositis, Volkmann's contracture. They are especially valuable, apparently, in sciatica and lumbago. Diathermy may be used to warm paralyzed limbs that are cold and cyanotic. The vacuum electrode has been found useful in trench feet.

The static current is not as a rule available in military practice as the machine is cumbersome and is sensitive to dampness, therefore is difficult to keep in good condition.

Phoresis and ionization.—The power of the poles of the galvanic current to cause diffusion of various substances from them and to cause them to pass through the skin has been utilized in medicine. The former is known as ionization or ionic medication, the latter as phoresis.

Iodin and salicylic acid may be so used in the treatment of arthritis, neuritis, and injured nerves. Zinc ionization has been recommended in indolent ulcers and wounds. Foul, indolent wounds

have been favorably influenced, it is said, by the introduction of chlorin by this method.

Mechanotherapy.—Many forms of mechanical apparatus designed to bring into play various groups of disabled muscles and thus increase their functioning capacity have been devised. While some of the simple forms are of distinct advantage, the results obtained have not justified the creation of an extensive equipment because of the difficulty in keeping up the patient's interest, and inducing him to give them the constant use required to make them serve their intended purpose.

Far better results have been obtained by expansion of the principles of *curative workshops* described below.

Curative workshops.—Very important among these types of treatment is that of the curative workshop training as distinguished from the purely vocational training which belongs to a later period and aims at such different ends and by such dissimilar methods that it should be kept apart from the former in time if not in place as well. The former should be instituted at the earliest moment physically possible, often even while the patient is still a bed patient; the latter only when the former in connection with other purely therapeutic measures has restored as fully as is possible all the functions of the members lost or impaired by the injury.

The aim of the therapeutic or curative workshop treatment is, roughly, three-fold:

1. To keep the mind of the patient occupied by interesting work during the many months of convalescence, especially at first when he is prone, otherwise, to spend his time pondering over his hopeless, helpless lot.

2. To restore the habit of work which is so frequently lost during the period of suffering and discouragement associated with any long course of hospital treatment.

3. To obtain the greatest possible range of usefulness to structures directly or indirectly affected by the injury. In other words, both mental and physical reconstruction.

For these reasons, curative workshop treatment should be instituted in a modified form as soon as operative procedures, splints, and dressings permit.

Certain types of work can be carried out in bed if a hand or hands are available in any form. A few hours daily on a piece of embroidery or knitting which is of interest to the patient will accomplish infinitely more in preserving or restoring function of the fingers or wrist than the routine use of various forms of mechanotherapy which involves the use of an elaborate machine taken in rotation for a few moments each by a series of patients.

The shop should be furnished with electric power and should be divided into sections to give a reasonable variety of occupations. Whereas some power-driven machinery, such as lathes, drills, etc., are advisable in order to make the production of metal splints and other hospital equipment possible, the simpler the workshop tools and apparatus and the easier their use can be grasped by the patient, the better. Carpentering, cobbling, leather working, cabinet work, tailoring, wood-carving, fretsaw, leather embossing, metal work (especially for apparatus and splints), printing, weaving, basket making, as well as typewriting and bookkeeping represent some of the most satisfactory types of work.

The surgeon in charge of the patient should prescribe the general type of exercise desired to be obtained in the workshop, based on the condition of the structures involved. The administrator of the workshops will order the most suitable type of work to obtain these results, varying the type of work in accordance with continued observation of the patient's mental and physical capacity.

Whenever individuals skilled in any particular occupation are found among the patients, whose general treatment will continue over a long period, they should be used as instructors in the workshops, supplementing the civilian instructors. A certain number can be used for clerical work. Non-commissioned officers who are patients, frequently are very useful when put in charge of the men in workshops in charge of matters of discipline.

During the period of workshop treatment the importance of other treatment ordered by the surgeon in charge of the case, such as massage, electro-therapy, etc., should not be overlooked, and appointed hours for such treatment should be arranged to fit in with the workshop hours in such a way as to cause as little delay and confusion as possible.

The curative workshop treatment should continue as long as there is possibility of any marked physical improvement from such treatment. During this period the case should be studied by the vocational director with a view to the permanent vocation to be recommended, and in some cases the curative work can be directed toward that end; but the purely vocational treatment belongs to the following stage of the treatment of the case, when facility and capacity for the special line chosen can be developed freely.

This following stage the formerly helpless, disabled, and discouraged soldier can now approach with a feeling of confidence and a hope in the future which would have seemed impossible before the curative workshop treatment was first undertaken.

CHAPTER X.

THE SYMPATHETIC SYSTEM.

The sympathetic system differs from that already described in being particularly concerned in carrying efferent and afferent impulses to and from the thoracic and abdominal organs in contrast to the great somatic masses of voluntary muscle. The efferent or motor paths are peculiar in that they supply the involuntary and cardiac muscle and the glandular tissue and also because they consist of at least two links between the source of the impulse (the spinal cord) and the structure to which it is distributed. It is these links that constitute the sympathetic elements proper—the *sympathetic neurons*. The cell-bodies of these neurons exhibit a disposition to become aggregated into larger or smaller collections, which constitute the *ganglia* that form a conspicuous feature of the sympathetic system. The sympathetic system consists of a complex of spinal and sympathetic fibers intermingled with groups of ganglion cells.

Constitution and arrangement.—The sympathetic system comprises two principal parts, the *gangliated cords* and the *plexuses* with their associated *ganglia*.

The gangliated cord, one of a symmetrically placed pair of gangliated trunks situated anterior or lateral to the bodies of the vertebræ, begins in the head and extends through the neck, thorax and abdomen to the lower portion of the pelvis. In the head it consists of a plexus of fibers continued up from the neck in an intricate interlacement which follows the internal carotid artery; and in the pelvis it terminates by the two cords forming a loop situated anterior to the coccyx and containing the *coccygeal ganglion* or *ganglion impar*.

The plexuses are a series of more or less distinct collections of nerve-cells and fibers, situated mainly in the axial line and giving off and receiving fibers connected with the various viscera.

The ganglia, although following a general plan of arrangement as to number and position, are subject to wide variations. A ganglion may or may not be connected with a spinal nerve, but it is always linked by *association cords* with other ganglia. According to their position, three varieties of ganglia are recognized. One group includes the *prevertebral ganglia*, those found as nodes in the gangliated cord; a second variety comprises the *collateral* or *intermediate ganglia*, which lie either on the peripheral branches of the gangliated

cord or in a prevertebral plexus; to the third set belong the minute *terminal ganglia*, composed of nerve-cells which lie at or near the visceral distributions of the sympathetic fibers.

The nerve-fibers in the sympathetic system include two sets, those derived from the cerebro-spinal system, which are medullated, and the sympathetic fibers proper, for the most part non-medullated.

Rami communicantes.—Where the typical segmental arrangement in the ganglionated cord prevails, as in the thoracic region, each spinal nerve is connected with the adjacent ganglion by a pair of short nerve-trunks, known as the rami communicantes. These are divided into two groups, the *white rami* and the *gray rami*, a distinction depending upon the difference in the appearance of the strands when seen in the fresh conditions.

The white rami communicantes are composed of the visceral branches of certain of the spinal nerves which use the sympathetic system as the pathway by which they arrive at their destination. They consist of medullated nerve-fibers derived from both the anterior and the posterior roots of the spinal nerves. Not all of the spinal nerves give off white rami, these strands of communication forming a *thoraco-lumbar group*, from the first or second thoracic to the second or third lumbar nerve inclusive, and a *sacral group*, derived from the second and third, or third and fourth sacral nerves. The cervical nerves do not give off white rami.

The gray rami communicantes are bundles of sympathetic neurons which pass from the gangliated cord to each one of the entire series of spinal nerves. The reason of this provision will be evident when the purpose of the communications effected by the gray rami is recalled, namely, to provide sympathetic filaments to the outlying muscles and glands by way of the path afforded by the distribution of the somatic nerves.

The further course of the sympathetic fibers, after having joined the spinal nerves by way of the gray rami, is as follows: They may course along with the anterior or posterior divisions of the spinal nerve and convey vasomotor, pilomotor, or secretory impulses to the involuntary muscle and glands, or they may enter the spinal canal by way of the anterior or posterior nerve-roots and be distributed to the spinal meninges.

The association cords are the longitudinally disposed bundles of fibers comprising the interganglionic portion of the gangliated cord; they contain both white and gray fibers.

The branches of distribution from the gangliated cord include the *somatic* and the *visceral*. The somatic branches are the rami communicantes; the visceral branches comprise the splanchnic efferents, which consist of both white and gray efferent fibers, as well as the white splanchnic afferents.

Relations of the sympathetic and autonomic divisions.—Anatomically, it appears that two types of physiological activity are present in the vegetative nervous system. These have been termed the sympathetic

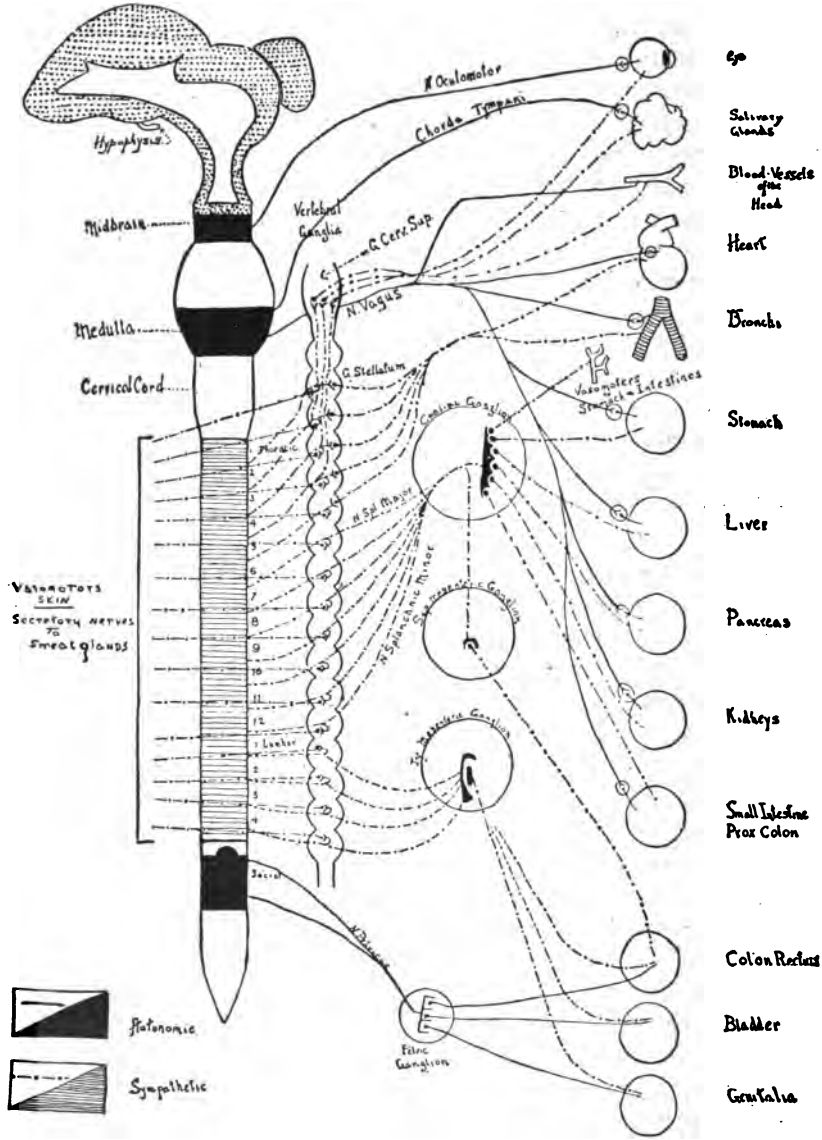


FIG. 206.—Distribution of the various divisions of the vegetative system. Sympathetic fibers are indicated by dotted lines; parasympathetic (autonomic) by unbroken lines. (Kraus, modified from Meyer and Gottlieb, *Jour. Nervous and Mental Disease*, 1917.)

and the parasympathetic or autonomic. All these non-voluntarily influenced organs, smooth muscle structures, heart muscle, glands, whose nerve fibers are derived from the spinal cord from the first dorsal above to the fourth lumbar segment below, belong in the sym-

pathetic system in the narrower sense. All others are controlled through the parasympathetic or autonomic. The uppermost come from the midbrain, enter the ciliary ganglion, and are distributed to the smooth internal muscles of the eye. A second or bulbar autonomic system passes through the facial and goes as the chorda tympani to the salivary glands. The glossopharyngeus and vagus belong to this bulbar autonomic system. A sacral autonomic system supplies the organs of the pelvis and genitalia. (Fig. 205.)

These two systems, while mutually antagonistic in their muscular effects, are mutually cooperative in maintaining a physioligical balance in the parts innervated by them. Certain chemical substances have selective affinity for each of these two correlated systems. Thus, some substances are sympathetico-tonic. Other substances, affecting the chief representative of the autonomic system, the vagus, are vagoparalytic, while others are vagotonic.

Disease of the sympathetic and autonomic systems lead to three major groups of clinical disorders: (1) Angio-neuroses; (2) tropho-neuroses, and (3) organo-neuroses.

The angio-neuroses.—Of the angiomotor neuroses some result in a hyperemia of the parts affected, others in an ischemia or anemia. In both instances, the disturbance is due to a sympatheticotonic condition, or prolonged sympathetic stimulation.

1. *Erythromelalgia* or *Mitchell's disease* may be mentioned as representing the hyperemic type of sympatheticotonia affecting the blood vessels. This is a paroxysmal disorder marked by painful redness and swelling of the feet.

Course and treatment.—The outcome depends much upon the causation. An erythromelalgia due to spinal changes (syphilis, tumor, multiple sclerosis) may get better if these improve. The therapy will be determined by the cause. Palliatives, such as the use of high-frequency current, violet rays, cold, antipyrin, are valuable as well.

2. *Raynaud's disease*, symmetrical gangrene or local asphyxia, is an instance of the ischemic type of sympatheticotonia affecting the blood vessels.

Treatment.—Therapy is often without avail, as the underlying condition is unmodifiable (syringomyelia, multiple sclerosis, etc.). As a rule, however, the attack subsides, although to appear again. Then attention should be addressed to the general health of the patient, especially to emotional features which produce vascular instability. Mild massage, local warmth and Bier's hyperemic treatment are of value during the attack, strong analgesics being necessary for the pain at the time.

3. *Intermittent claudication* is an angiospastic syndrome characterized by a limp, with weakness, pain, and coldness in the affected limb.

Treatment.—Rest, warm applications to the parts, and high-frequency current are of value in treating the attack in its acute stage. Treatment of the condition rests upon the proper conception of the individual provocative disorder. Arteriosclerotic cases need treatment for this; psychoneurotics require psychotherapy.

The tropho-neuroses.—These conditions are represented by the trophic dermatoses and certain trophic disorders of the bones and joints.

1. *Scleroderma* or hardening of the skin is one of the most prominent of the tropho-neuroses. Its etiology is little understood, but its pathology seems to have its basis in an increased fibrosis due largely to imbalance in the sympathetic system.

Treatment.—The therapy will depend upon the underlying factors. The endocrinous and psychogenic cases would seem to offer the most opportunity either by carefully balanced opotherapy or by psychotherapy.

2. *Multiple neurotic gangrene of the skin* is a dermal process in which vasomotor constriction is not limited to the extremities but appears in isolated patches of the skin.

3. *Arthropathic tropho-neuroses.*—Conditions of this type are represented in the well-known arthropathies of tabes dorsalis, i. e., the Charcot joint.

The organo-neuroses.—(1) *Gastro-intestinal syndromes.*—In this group are to be included *tabetic gastric crises*, which are probably vagotonic disturbances and which consist of nausea, vomiting, hypersecretion, pain, and hyperesthetic skin-zones; *vagotonic manifestations* in the gastro-intestinal tract, although not yet completely analyzed, are nevertheless of much clinical importance. These disorders include esophageal cardiac spasms, sialorrhea, retarded peristalsis, hypersecretion, hyperacidity accompanied by pylorospasm. Membranous colitis or enteritis, nausea and vomiting are often associated with the condition referred to as *vagotonia.*

(2) *Genito-urinary syndromes.*—A number of functional disorders are recognized in connection with the urinary and sexual disorders, as for example, urinary urgency, nycturia, nocturnal enuresis, impotence, premature ejaculation, priapism, and sexual crises. Undoubtedly, although many of these conditions, like all of the organo-neuroses, have a large representation upon the psychic level, they do, nevertheless, depend in no small measure upon disorders of the sympathetic and autonomic systems.

(3) *Cardiac syndromes.*—In this category are included those symptoms due to functional disorders of heart action, such as bradycardia, tachycardia, arrhythmia, and pseudoangina. The psychic elements in these disorders are easily demonstrated, but they make

themselves apparent in an imbalance between the autonomic and sympathetic systems.

(4) *Respiratory syndromes.*—In this group are classed such disturbances of respiration as appear to have a wholly functional basis, such, for example, as asthmatic attacks with spasm of the bronchi, slowing of respiratory phases, pseudo-dyspnea, and emphysema, all of which are seen in vagotonia.

(5) *Ocular syndromes.*—The ciliary, pupillary, sphincter, and dilator muscles, Müller's orbital muscles, and the tear glands are all organs of the eye which are innervated in part by autonomic and in part by sympathetic fibers. Several important clinical conditions depend upon the complicated pupillary innervation, the chief of which are absolutely stiff pupils and sympathetic paralysis.

Sympathetic ocular paralysis, also known as Horner's syndrome, is characterized by retraction of the eyeball, narrowing of the palpebral fissure, drooping of the upper lid, elevation of the lower lid, myosis, and reduction of the psychical and light reflexes of the pupil. This clinical picture is due to pressure upon the sympathetic fibers.

The complete loss of all pupillary movement, producing absolute pupillary rigidity, is seen particularly in fainting, high grades of anxiety or fear, hysteria, in most epileptic seizures, and in cerebrospinal syphilis.

The nature of endocrine disturbances.—Endocrine disturbances, that is, interference of the mutually balancing units of the internal glandular system, may be brought about in a variety of ways. If we consider the normal relationship existing between these units, some of the basic disturbances become clear to us. Thus, the pineal and thymus glands are supposed to cease their function and gradually involute at puberty.

If the *pineal* ceases before this time, then the inhibition it exerts on the gonads also ceases and allows these latter to develop prematurely, and precocious puberty results with all its attendant symptoms and signs. The genitals enlarge, pubic hair makes its appearance, early initiative and mental precocity arise in the individual. But these advances are made at the expense of the normal structural development, and hence smallness of stature and weakness of musculature may accompany the sexual characteristics.

If the *thymus* involutes too early in life, then the bony structures of the body suffer. Calcium metabolism is interfered with, the bony framework remains small and weak besides, with distortions and deficiencies. The reaction of the system to toxic invasion is diminished, and convalescence is long continued and interrupted. Too long-continued activity on the part of the pineal after the age of puberty results in the gonodal development being held in abeyance.

The genitals remain small, although the stature increases; pubic hair remains scant, and the mental reactions remain childish. The conformation of the body is that of an overgrown child, adiposities and rounded-out joints being prevalent. When thymus involution is retarded in adolescence we have also striking changes. The individual keeps on growing, albeit the bony structure is wanting in strength, and the sex glands likewise are held in abeyance. The skin retains the characteristics of childhood, being soft and smooth and velvet-like, with the fine, downy hair (lanugo) seen in the young. If such individual is a male, he almost never shaves and he has the complexion of a girl. A girl with thymic sub-involution menstruates late in adolescence and quite irregularly. These cases of thymic subinvolution have for us a very great importance apart from these functional and structural anomalies. It is found that their coefficient of carbon-dioxid combining power of the blood pressure is low, and hence they are subject to a condition of acidosis on very slight provocation. This makes them bad operative risks and makes them prone to serious complications in any infectious disease. Furthermore, they are usually found to have a very small amount of available adrenalin, and hence overactivity, especially intensive muscular exertion, frequently ends in sudden death. They have what is known as "suprarenal heart"—no murmur—but profound and rapid exhaustibility. Their skeletal musculature also is rapidly exhausted, and extreme fatigability ensues.

Furthermore, as the thymus is an autonomic irritant, all the symptoms of vagotonia are found exhibited to a greater or less degree in such individuals, namely, asthmatic attacks, gastric pain with pyloro-spasm, abdominal disturbances, slow pulse with low blood pressure, profuse sweating, atonic bladder walls resulting in enuresis, and so on.

The three glands, *thyroid*, *suprarenal*, and *pituitary*, are extremely closely related, especially in their effect on sugar mobilization, on growth, on blood pressure maintenance, and on their reciprocal activity upon the autonomic and sympathetic divisions of the vegetative nervous system. Thus it is fairly well established that the thyroid secretion sensitizes the neuro-muscular synapse to the action of adrenalin. A deficiency in the thyroid, therefore, would make secondarily for a lowered tone of smooth muscle fiber, with lowered arterial tension. In such cases, the pituitary gland frequently seems to take on some of the qualities of the thyroid, and such pituitaries occasionally contain colloid material analogous to that of the thyroid.

The *thyroid* gland also controls the further disposition of the amino-acids produced by protein metabolism in the body and prepares them for final excretion. If this process is held in abeyance, protein metabolism diminishes, and the partial products are retained

by the organism. This lack of oxidation, together with the lowered blood pressure and diminished pulse rate due to lack of sensitization for adrenalin, gives the clinical picture seen in myxedema. Hereto must be added mental torpor and body sluggishness. An excessively acting thyroid, on the contrary, with its great pull on the protein constituents of the body cells, causes rapid oxidation, heightened blood pressure (if the adrenals are not deficient), rapid pulse, and increased body temperature. This makes for a loss in weight, great activity, both mental and bodily, extremely heightened reflexes, and the rapid production of fatigue seen in hyper-thyroid states.

The *pituitary gland* has a variety of functions to perform. Its anterior lobe has been said to stimulate bodily growth, and the active principle of such growth has lately been isolated by Robertson, of Berkeley, Cal., and called by him "tethelin." Its function seems to include also that of sex gland stimulation. The posterior lobe is said to contain no less than eight active principles which individually act, first, to maintain blood pressure not only through the agency of a pressor principle, but also of a depressor one, which has been extracted from the gland with alcohol; second, to increase peristalsis and stimulation of the sympathetic system generally, thereby improving nutrition; third, to increase the production and flow of the mammary glands; fourth, to stimulate uterine contractions; fifth, to increase urinary secretions; sixth, to increase the absorption of cerebro-spinal fluid; seventh, to stimulate metabolism; and, eighth, on account of the stimulating properties of the sympathetic, the posterior lobe has become a valuable remedy in surgical shock, far more efficacious than adrenalin.

Its increased activity is usually coincident with diminished sugar tolerance, and one of the methods of determining such hyperactivity is by the relative amount of sugar that can be taken at one time by the individual without sugar appearing in his urine. The normal amount of sugar toleration is 2 grams per kilogram of body weight. If much more than this can be tolerated, we presuppose a hypo-activity of the posterior lobe. It is interesting to observe that all the periodic functions of life seem to run parallel with pituitary activity. Yearly hibernation in the woodchuck, as shown by Cushing, is synchronous with a diminution in the size of the gland. Many cases of narcolepsy—that is, attacks of sleep—coming on at intervals without known cause, seem to be due to disturbances in the pituitary. The periodicity of the menstrual function likewise is attributable to a normally acting pituitary gland. An increase of its secretion produces a lessening of the inter-menstrual period. From the variety of well established functions of this gland, one can readily picture the disturbances that may arise from its dysfunction. Apart from

the consideration of functional disturbance, its topographical relations to adjacent structures must be a factor, when enlarged, in the production of pressure chiefly upon the optic chiasm, but also upon the third, fourth, and sixth cranial nerves, the crura cerebri, and the neighboring temporo-sphenoidal lobes of the cerebral hemispheres. Either the purely glandular or the pressure symptoms may usher in the clinical picture, and while the functional disturbances of growth and metabolism are almost invariably present in the completed syndrome, yet the symptoms referable to pressure on neighboring structures need not be. It is only in marked hyperplasias or neoplasms that these pressure symptoms arise. If the enlargement is proceeding anteriorly, then the optic chiasm is involved, and various degrees of temporal hemianopsia result:—unilateral or bilateral or perhaps only scotomata. If the mass presses laterally, we get progressive involvement of the nerves adjacent to the cavernous sinus—the third, fourth, and sixth cranial—and finally an obstruction to the sinus circulation. These conditions produce various disturbances in the movements of the eyeball, with ptosis, strabismus, diplopia and gradual loss of vision. Occasionally, choked disc is produced. If the enlargement exerts pressure backward, either one or both of the crura cerebri may be affected, and all the signs of pyramidal tract involvement supervene, with exaggerated reflexes, Babinski sign, spasticity. Lateral or postero-lateral growth irritates the surface of the temporo-sphenoidal lobe of the cerebral hemisphere, and such cases frequently have epileptoid seizures with auræ of taste—uncinate fits.

From the foregoing it will readily be appreciated how varied and complicated may be the picture produced by enlargements of the pituitary body. Röntgenology offers some help in these cases. There is deepening and widening of the sella turcica, with absorption of the posterior clinoid processes.

In a large percentage of cases pituitary disorders require operative interference, at least for the preservation of vision; but there are cases now on record in which a bitemporal hemianopsia, headaches, and other conditions of functional disorder have disappeared under the administration of pituitary substance or pituitrin with or without thyroid extract.

The *suprarenal* glands are composed of two distinct parts which are functionally widely different in their effect—the medulla and the cortex. The proportion between them in man is as one to nine; that is, the cortex is nine times the volume of the medulla, and forms nine-tenths of the entire gland. The medulla produces adrenalin and the cortex some active principle which as yet has not been isolated, but which controls to an extent the sexual characteristics of the individual. At least, we know that tumors or hemorrhages into

the cortex are followed by cessation or diminution of menstruation, by the masculine type of pubic hair, by the growth of mustache and beard in the female. This is not so surprising when it is realized that phylogenetically the ovaries and the suprarenal cortex arise from the same tissue in the embryo. In some lower forms, indeed, cortex and medulla are quite distinct, and form separate organs. It is supposed that the active principle of the cortex stimulates the autonomic portion of the vegetative nervous system as contrasted with the adrenalin of the medulla, which is known to stimulate the sympathetic division. It is a mistake to assume that the suprarenal medulla alone produces adrenalin. This substance is secreted by cells of the chromaffin system throughout the body and notably in the large groupings of such cells found at the carotid angle and at the bifurcation of the aorta. Experiments made upon suppositious deprivation of adrenalin by removal of the suprarenals are not, therefore, conclusive nor sufficiently exclusive and must be interpreted accordingly.

Most observers are agreed that adrenalin is the great activator of the sympathetic system. Under its influence the tone of smooth muscle fiber is maintained throughout the body. This includes arterial tension and is especially important as far as it relates to the abdominal blood vessels, for in all cases of shock from whatever cause, the accumulation of blood in these vessels is of prime importance. Such a condition may be brought about by rapid diminution or destruction of the available adrenalin of the body, and among the most potent means to combat it is the introduction of adrenalin into the circulation. Of late it has been found that pituitrin is also a specific for restoring tone and pressure, and its effect is more lasting. A curious exception to the action of adrenalin on the blood vessel walls is furnished by the arterioles of the pulmonary system, those of the cerebrospinal system, and of the coronary arteries. Here adrenalin is practically inert.

Another important property of adrenalin is in its effect upon sugar mobilization. Injections of adrenalin or stimulation of the splanchnic nerves which activate the suprarenals, produce on the liver an effect that releases the stored-up glycogen for immediate use by the muscular tissue of the body. Thus increased muscular activity is made possible. The posterior lobe of the pituitary also is productive, as we have seen, of an increased amount of blood sugar, but this possibly arises from its antagonistic action upon the pancreas, inhibiting the latter from properly controlling the sugar intake. The thyroid gland also is instrumental in producing an excess of circulating sugar. This action is presumably due to sensitization of the tissues for adrenalin—an effect of thyroid administration which of late has been more or less recognized. The three glands—thyroid,

adrenals, and pituitary—therefore all bear part of the burden in periods of great strain and stress, especially in the matter of blood pressure in muscular activity and in the prevention of shock.

The scope of this discussion does not permit of any further elaboration of the endocrine theories, but perhaps sufficient has been said to form a basis for further study, especially from the clinical side.

Appended hereto is a table which classifies the chief changes in the various tissues of the body due to endocrine imbalance. It is hoped that this table may be used as a field chart for the ready detection of the more pronounced clinical features of such disturbances.

TABLE VIII.—*Determination of endocrine disturbances.*

Tissue.	Disturbance.	Check.	Chief gland affected.
Skeleton	*Height abnormal:*		
	(*a*) Over 6 feet		Pituitary.
	(*b*) Under 5 feet 5 inches		
	Abnormality in size and shape of skull		
	Kyphosis		
	Disproportion between size of extremities and trunk		
	Prognathism of upper jaw		
	Prognathism of lower jaw		
	Broad jaw with spaced teeth		
	Narrow jaw with crowded teeth		
	Feminine contour		
	Broad pelvis		
	Abnormal size of hands and feet:		
	(*a*) Too large		
	(*b*) Too small		
Skin	*Dry scaly skin*		
	Oily skin		Thyroid.
	Myxedematous skin		
	Complexion of infant with downy hair on lip and chin		Thymus.
	Brittle nails		Thyroid.
	Urticaria		
	Dermographia		
	Reaction to stroking of skin of abdomen:		
	(*a*) *Red line with broad white border*		
	(*b*) *White line*		Suprarenal.
	Trophic eruptions of extremities		
Hair	Deficiency of hair on lips and chin		Thymus.
	Deficiency of hair on body generally		Thyroid.
	Excessive hair on body		
	Nasal brow (meeting of eyebrows across nose)		Pituitary.
	Abnormal distribution of hair on back or in sacral region.		
	Brittle or coarse hair		Thyroid.
	Feminine distribution of hair on pubes (i. e., horizontal upper margin of pubic hair).		Suprarenal-cortex.
Muscle	Skeleton sparsely covered with muscular mantle		Pituitary.
	Muscle dystrophy		Pineal.
	Rapid muscle fatigability (setting up exercise)		Suprarenal.
	Muscle and tendon contractures		Pineal.
Glands	Tonsils large		Thymus.
	Axillary and inguinal glands		
	Thyroid enlargement		Thyroid.
	Breasts present		Suprarenal-cortex.
Pigment	*Pigment in mucous membranes* (mouth)		do
	Pigment spots in teeth		
	Pigment in abnormal situations of body especially at site of pressure of clothes.		
Genito-urinary organs.	Polyuria		Pituitary.
	Malformation of sex organs		
	Undescended testicles		
Cardio-vascular system.	*Blood pressure:*		
	(*a*) *Over 150*		do
	(*b*) *Under 120*		Suprarenals.
	Pulse (at rest, standing):		
	(*a*) *Over 85*		Thyroid.
	(*b*) *Under 60*		
	Heart, muscular action inefficient		Suprarenals.
	Bluish extremities		do
	Blanching of digits of hand or foot		
	Mottling of skin		

CHAPTER XI.

THE MENINGES AND THE SPINAL FLUID.

ANATOMICAL CONSIDERATIONS.

Before taking up a consideration of meningitis and its symptoms it is necessary to make brief mention of the anatomy and physiology of the membranes of the central nervous system. The customary descriptions deal with three definitive, well-separated structures—dura mater, arachnoidea, and pia mater. While such a division of these meninges into three parts is arbitrary, it seems best to continue the use of the terms, because of the possible simplifying of anatomical description.

Dura mater.—The dura mater is a strong, thick, and dense membrane, inclosing the central nervous system and constituting the outermost of the three meninges. Structurally it is composed of white fibrous connective tissue, with but few elastic fibrils—a tough, unyielding membrane. The cranial portion is usually looked upon as being composed of two layers; these split to inclose the large venous sinuses and certain other structures (such as the cavum Meckelii). In the spinal region these two portions are not in approximation, for the outer layer here serves as the periosteum of the interior of the vertebral canal (particularly on the anterior side), while the inner fraction becomes the true spinal dura. Between these two so-called layers of the dura in the vertebral canal occurs the epidural space, filled with fatty tissues and a large venous plexus.

Within the cranium, the dura adheres closely to the inner surface of the bones forming the skull cavity, serving there as the internal periosteum. The attachment to the skull varies greatly in intimacy in the different areas, for over the vault the dura can be stripped away fairly easily, while in the base, the connections are, as a rule, more resistant and more fixed, particularly along the line of the sphenoidal wing. This firmer attachment of the dura to the base of the skull is of importance in fractures; in the linear variety in this region the dura is practically always torn. Histologically, there is no separation of the dura into two layers, for the dense fibrous structure is everywhere similar. Hence the dura may be considered as a unit—a tough unyielding membrane lining the cranial cavity and covering the central nervous system.

In young persons particularly, the relation of the dura to the inner surface of the cranium is of interest. In infants before the closure of the fontanelles, the dura is intimately associated with the fibrous membrane closing the osseous gap. In the outer layer of this thickened fibrous membrane, bone is deposited by ingrowth from the cranial plates, the process ultimately closing the skull. This fusion accounts for the close attachment of dura to all of the suture lines in the young, particularly to those lines where growth is actively proceeding. It also gives an anatomical basis for the occurrence of isolated extra-dural hemorrhages in the new-born ("internal cephalohematoma") where bleeding is confined between the dura and the inner surface of the single bone.

The dura completely incloses the central nervous system as an intact membrane. It extends outward along each of the spinal nerves terminating on the distal side of each root ganglion. In the cranium a similar extension occurs in the case of the more caudal cerebral nerves, but with the others (particularly the optic) the relations are somewhat different. Elsewhere, this dural cuff, as it loses its identity peripheral to each ganglion, continues outward somewhat by blending with the external perineurilemma of each cerebrospinal nerve.

The dense fibrous character of the pachymeninx causes it to play an all-important part in the establishment of the *indistensible character* of the cerebro-spinal spaces. The space inclosed by the dura is almost absolutely fixed in capacity in adult life, this membrane providing for practically no enlargement of the fluid capacity. The dura must be assumed to be the chief factor in this "closed cavity," for the bony cranium adds support only to a portion of the whole cerebrospinal space. The intracranial extensions of the dura (*falx cerebri* and *tentorium cerebelli*) also act as inelastic supports; the tentorium surely protects the posterior chamber from much of the direct pressure exerted from above. The importance of the inelastic dura must be realized by all cranial surgeons, for cerebral decompressions without wide opening of the dura provide no relief for increased intracranial pressures.

In younger persons, especially in infants, the dura mater may be more readily distended than in adults. This is shown by the slow but very marked increase in the cranial vault in cases of internal hydrocephalus, in which enlargement of the intradural capacity must accompany increased aggregation of fluid. In adults an acutely developing abscess or local meningitis of one hemisphere may dislocate considerably the falx cerebri, rendering the whole brain asymmetrical. It seems also reasonable to suppose that abnormally high pressure exerted for a longer period may occasion local or general dislocations of the tentorium, permitting cerebral growths to press upon cerebellum, and so forth. But the extent of this dis-

location in adults is not very great; the falx tends to protect one hemisphere from the other, and the tentorium isolates almost completely the essential bulbar portion of the brain stem from injury from above.

The inner surface of the dura is covered by a single layer of polygonal mesothelial cells. These cells form a fluid-containing layer for the outer surface of the subdural space. Surgically they are interesting in their relations to adhesions between dura and arachnoid; before such a joining of tissues can take place, injury to these flattened cells probably must occur. Whether the injury must extend to the adjacent arachnoid elements is uncertain; it suffices to assume that injury to the cellular linings of the subdural space permits adhesions between dura and arachnoid.

Blood vessels.—The chief vascular supply of the dura is from the middle meningeal artery, the only vessel of surgical importance in this membrane. This vessel arises from the internal maxillary branch of the external carotid artery and enters the skull through the foramen spinosum. Its further course is an extension over the lateral hemisphere between dura and bone, often grooving the latter deeply. The mechanical separation of the dura from the inside of the cranial vault frequently results in rupture of this vessel—a troublesome occurrence during an operation and a complication in certain cranial fractures. The artery occasionally must be dealt with in approaches to the Gasserian ganglion requiring section between ligatures close to the base of the cranium, or, in more difficult cases, plugging of its foramen.

But the chief interest in the blood vessels of the dura mater concerns the peculiar *venous sinuses*. These large channels lie within the dura itself—a splitting of this dense membrane to compose the vessel walls. In consequence the histological structure of these sinuses is characteristic; the walls are made up almost entirely of white fibrous connective tissue (with but a small admixture of elastic elements) and are entirely lacking in muscle fibers. The channels are, however, completely lined by typical vascular endothelium. In consequence of this peculiarity of structure, the dural sinuses may be assumed to possess no power of contractility. Variations in the volume of flow through these vessels must occur, then, in response to alterations in the rate of the flow and not because of active changes in the lumina.

The protected character and fibrous architecture of the venous sinuses of the dura have caused most investigators to regard them as incompressible. Cushing's experiments upon the subject of local and general cerebral compression demonstrated 15 years ago that in the dog a total collapse of the superior sagittal sinus might be

occasioned during an early stage of compression. Thus, changes in the lumen of the great sagittal sinus (and probably of the others) may be produced passively and not actively. Whether this phenomenon of compression of the dural sinuses plays a great rôle in the normal adjustments of the cerebral venous drainage and of the intracranial fluid-distribution can not be told.

Of these pachymeningeal channels, the *superior sagittal sinus* probably possesses the most interesting peculiarities. It extends from the ethmoid region posteriorly in a great curve, lying in and forming a part of the prismatic line of attachment of the falx cerebri to the dura of the calvarium. On cross section the lumen is triangular, the base being the dural attachment and the two sides being formed by the division of the fibers of the falx. The lumen of the sinus increases gradually as the torcular region is approached, but in the middle extent this channel widens broadly into the *lacunæ laterales*. These are lateral diverticula of the sinus, stretching out over the precentral region for about 2 cm., as interrupted, tortuous but communicating venous lakes, endothelial-lined and receiving into their channels the blood from the chief of the superior cerebral veins. Communications between the dural sinuses and the diploetic vessels may occur in this region. Into the lacunae laterales project the greater number of the arachnoid villi, about which more will be written in the discussion of the leptomeninges.

The great sagittal sinus, particularly in the regions of its lateral expansions, is characterized also by the occurrence within its lumen of many fibrous, endothelial-covered bands, the *chordæ Willisii*. These usually extend from one wall to the other, in the prismatic portion of the sinus. The occurrence of these bands in the sinus and of similar, yet larger and more complicated columns in the *lacunæ laterales* suggests that these represent a persistence of the pattern of the embyronic plexus.

At the junction of the falx with the tentorium, the great longitudinal sinus opens into the *confluens sinuum* (torcular Herophilii). Here is collected the blood from both of the sagittal sinuses (the inferior lying along the free border of the falx and uniting with the great cerebral vein of Galen to form the unpaired sinus rectus), and from this point the venous drainage is continued downward by the two *transverse sinuses*. These curve laterally in the zone of tentorial attachment around upon the mastoid portion of the skull. In this area the sinus usually receives a large emissary vein from the diploe. From the mastoid region, the transverse sinuses continue in a sigmoid curve downward to form at the jugular foramina the two internal jugular veins. The sinuses may be exposed in their middle course without marked hemorrhage, but in the region of the occipital

protuberance (near the torcular) emissary veins form a complicating factor.

Into the first part of the transverse sinuses open the *occipital sinuses;* or, if these two occipital structures have united, the single vessel opens into either right or left transverse sinus. These occipital sinuses lie in the attached border of the falx cerebelli and in the dura of the posterior lateral border of the foramen magnum. They form anastomosing channels between the upper and lower portions of the tranverse sinuses and they communicate with the posterior spinal veins. They are quite variable and may prove troublesome in operative approaches to the cerebellum.

In addition to this dorsal system of venous sinuses, there is another important series of large venous channels in the basilar portion of the skull. On both sides of the sella turcica occur the symmetrically placed *cavernous sinuses*, receiving the drainage of the orbit and lying in close approximation to the entering internal carotid arteries. These cavernous sinuses communicate across the midline through anterior and posterior sellar plexuses, forming a circular sinus in the dura surrounding the hypophysis cerebri. The chief surgical significance of these cavernous sinuses lies in the likelihood of their involvement in fractures of the base, and also in the possible formation of an arterio-venous anastomosis between the internal carotid and the sinus. On thrombosis of these sinuses an extreme exophthalmos is produced.

Drainage of blood from the cavernous sinuses may be intra-cranial or extra-cranial. The former pathway is a double intra-dural channel by way of either the superior or inferior petrosal sinuses, emptying finally into the jugular vein. The much smaller extra-dural escape for the cavernous blood is effected through an emissary vein into the pterygoid plexus or into the angular vein by way of the superior ophthalmic. The petrosal sinuses, particularly the superior, are apt to be injured in fractures of the base of the skull, for in this petrous region the attachment of the dura to the cranium is exceptionally intimate.

The leptomeninges.—The more delicate membranes surrounding the central nervous system are the arachnoidea and the pia mater. These are, by their gross characters, sharply differentiated from the dura mater; they are thin, friable membranes, almost transparent, yet serving as efficient fluid-barriers. For the leptomeninges must be chiefly considered from the viewpoint of the circulation of the cerebrospinal fluid within them, a physiological as well as anatomical conception.

The outer of the two leptomeninges—the arachnoidea—is customarily described as a delicate membrane, of glistening exterior sur-

face, bridging the cerebral sulci and serving as a fluid-containing membrane. In the spinal region, the arachnoid is a sheath closely approximated to the dura. Such a picture of the arachnoidea is hardly complete, for it takes account of only the more obvious continuous character of this leptomeninx. In reality the structure should be considered as of two parts, the *arachnoid membrane* (the outer continuous cell-layer investing the whole central nervous system) and the *arachnoid trabeculæ* (the cellular columns which stretch from the inner surface of the arachnoid to the pia mater).

On this basis the arachnoid is a cellular structure with a delicate supporting reticulum of white fibrous and yellow elastic fibrils, covered on both sides by characteristic flat cells. From the inner side of this membrane project small, easily-broken trabeculæ; upon which are continued the cells forming the inner lining of the arachnoidea. The core of such an arachnoid trabecula is composed of a few connective tissue fibrils, united on the outside with the fibrous framework of the membrane and on the inner side with the sub-pial tissues. For these trabeculæ fuse with the reticular pia mater; the mesothelial cells, which compose the arachnoidal coverings, are continued upon or are identical with the cells of the pia mater. For this inner membrane of the nervous system is formed by the innermost reflection of the mesothelial cells; it constitutes the visceral (by analogy with the true serous cavities) layer of the subarachnoid spaces.

The *pia mater*, then, closely invests the nervous system, continuing into the cerebral sulci and into the medial fissures of the cord. Actually, as suggested above, it is essentially constituted by the "visceral" reflection of the cells of the arachnoidea, but it possesses in addition an underlying supporting structure of a few connective tissue fibrils and neuroglial elements. The exact relation of the superficial neuroglial elements to the pial membrane are as yet not wholly understood; it is quite possible that the neuroglial fibers add much to the basal reticulum of this investing layer.

An additional character of the pia concerns the vascular plexuses, usually described within this membrane. These vessels, both artery and vein, however, are beneath the essential layer of mesothelial cells in the pia, so that the complex vascular channels may be considered as subpial; the pia mater should hardly be termed the "vascular membrane of the brain." Quite similarly, all of these vessels perforating the fluid-spaces between pia and arachnoid are likewise covered by an inclosing investment of mesothelial cells. Thus, the subarachnoid space (or, better, spaces) becomes a fluid-bed, everywhere lined by the typical, very low mesothelial cells.

But the pia mater, unlike the arachnoidea, must not be considered as a wholly intact membrane without perforations. In places the membrane is seemingly incomplete. Thus, in the inferior velum,

the pia must be perforated if an actual medial *foramen of Magendie* exists (as seems likely), and likewise, a similar condition must hold in the rhombic lateral recessus, if the two *foramina of Luschka* are anatomical openings. If, on the other hand, these areas of fluid-passage are merely permeable membranes of differentiated ependyma, morphologically intact and complete, then surely the pia over these areas must assume the character of a similar, permeable membrane. Again, in relation to the perivascular channels, the pia seems perforated, in a special manner, by all the blood vessels as they enter or leave the nervous system. In these perivascular spaces, the pia enters as a mesothelial lining of the outer surface of the space; a variable distance from the surface of the nervous system, these cells become unrecognizable and are apparently lacking, replaced by neuroglial elements. The inner walls of these perivascular spaces seem likewise covered, for a certain distance, by mesothelial cells, reflected over the vessels from the arachnoid covering of all these vascular tubes as they traverse the subarachnoid spaces. Thus, while in general the customary description of the pia mater as a membrane closely investing the cerebrospinal axis and plunging into the cerebral sulci holds, the intact character of the pia as a cellular layer is probably incorrect.

The perivascular spaces must be looked upon as existing potential fluid-channels, which connect directly with the subarachnoid spaces. They offer a pathway of lesser resistance to the spread of infection from the meninges into the central nervous system, and are of great importance for this reason in pathological considerations of the nervous system. A perivascular infiltration is very common in acute meningitis; in poliomyelitis it is met with in association with the necrosis of nerve cells. Physiologically the perivascular spaces afford a channel for the fluid-elimination of the nervous tissue itself; evidence indicates that each nerve cell is surrounded by a potential fluid-space which drains outward into pericapillary and perivascular spaces into the subarachnoid spaces. Thus, an accessory fluid-system is afforded in the cerebrospinal axis by these perivascular channels; the drainage of waste products by this means is probably comparable to lymphatic drainage in other tissues, but within the central nervous system and its meninges true lymphatics are wholly lacking.

Thus, with the outlined arrangement of the three membranes about the cerebrospinal axis, two different fluid-spaces are formed. The outer of the two is the so-called *subdural space* enclosed between the dura mater and the arachnoidea. It is a very small space, the two limiting surfaces being separated merely by a capillary layer of fluid. Whether this fluid is exactly similar to the cerebrospinal fluid is difficult to ascertain. Likewise our knowl-

edge of the connections between the subdural and subarachnoid spaces is hardly definite. Hill (1896) believed the two spaces to be intimately connected, with easy fluid-exchange through foramina or by filtration. Quincke, many years before Hill, found an apparent passage of granular material only in the direction from subdural to subarachnoid. It seems established, however, that certain foreign salts in molecular solution, introduced into the spinal subarachnoid spaces, do not reach the subdural space either in the spinal or cranial regions. The subdural space has little of physiological or anatomical interest; the adhesion, however, between dura and arachnoid, with walling off of subdural spaces, is of great importance in perforating wounds of the head.

The second of the two spaces, the *subarachnoid space*, may, as judged from the foregoing account, be considered as merely an intra-leptomeningeal fluid-channel. For on the outer side the space is limited by the inner surface of the arachnoidea, while the inner wall is composed of the pia mater. The channels are interrupted, however, by the occurrence in them of definite arachnoid trabeculae, so that the fluid must traverse a devious but continuous passageway. Everywhere this intra-membranous fluid-bed is lined by the same type of cell, a low mesothelial element. These cells may become actively phagocytic and may assist greatly in the limitation of infection. In such circumstances, the cytoplasm usually becomes swollen and at times vacuolated. Certainly with the high colloidal dyes of the benzin series, this phenomenon of phagocytosis and swelling is present, as also in the case of particulate matter (carbon granules). But in infections the potency of the cells of the pia and of the arachnoid—these mesothelial elements—is remarkable in preventing the universal involvement of the nervous system (myelitis and encephalitis) and the spread outward into the subdural space.

Except in certain definite areas, the arachnoid membrane on its outer surface is entirely separated from the inner surface of the dura. The areas of fusion represent everywhere invasions of the pachymeninx by the arachnoidea. The most frequently found of these points of fusion are the *arachnoid villi*, prolongations of the arachnoid membrane into the dura so that the arachnoid mesothelial cells come to be directly beneath the vascular endothelium of the great dural sinuses. Arachnoid villi have been described in adult man, in the infant, and in the common laboratory mammals. Histologically these prolongations are covered by the typical arachnoid cells (low mesothelium), usually of only a single layer, but often forming whorls of many cells and presenting double-layered coverings. In these cellular whorls calcium may be deposited, forming the nucleus

for sand bodies (corpora arenacea) or starch bodies (corpora amylacea). Likewise these cell-aggregations may possibly be the initial growth-point of the so-called dural endotheliomata.

The core of these arachnoid villi is either a strand-like network, reduplicating the arachnoid spaces (as in the dog) or a myxomatous ground-work simulating the perimedullary mesenchyme. In addition to the true arachnoid villi (which occur in the walls of practically all of the dural sinuses), there are found infrequently projections of the arachnoid into the dura in other situations. Such arachnoidal attachments are sometimes seen at operations, stretching between arachnoid and dura; on being severed, clear cerebrospinal fluid escapes. From these projections, and especially from the villi, columns of arachnoid cells project varying distances into the dura. The arachnoid villi represent normal structures; the great enlargement of these in adult life in human beings (with frequent local excavation of the cranial vault) results in the formation of the well-known Pacchionian granulations.

The subarachnoid space, then, is a perfectly adapted fluid-channel within the leptomeninges, a cell-lined passageway for the circulating cerebrospinal fluid.

Ependyma and Choroid Plexuses.—The epidermal lining of the neural tube is termed the ependyma. This ependyma forms, in a layer of only a single cell in thickness, the inner investment of the whole central nervous system, the cells being of a cylindrical form with basal nuclei. Macroscopically the ependyma appears as a moist glistening membrane lining the cerebral ventricles; in the spinal region examinations in gross are unsatisfactory because of the small size of the central canal. The ependymal lining of the neural tube is intact and complete except in the region of the rhombic roof; here, as in the case of the pia, the foramina of Magendie and Luschka present obvious perforations.

Within the cerebral ventricles, the ependymal cells in certain locations undergo early differentiation and form the cellular covering of the vascular tufts (the choroid plexuses). Embryologically the first evidence of ependymal change appears in the roof of the fourth ventricle, at the early stage of about 15 mm. in man. The rhombic roof is divided by laterally developing invaginations of the ependymal elements, the tufts having a core of perimedullary undifferentiated mesenchyme. The complexity of the tufting increases rapidly, the cellular buds filling ultimately a definite portion of the ventricular space. Somewhat later (about 20 mm.), the choroid plexuses of the third ventricle make their appearance; the process of differentiation is here the same. Finally, the choroid plexuses stretch out into the lateral ventricles as well as the third and fourth. Grossly these

choroids are reddish, highly vascular, complex structures, occurring within the cerebral ventricles and possessing definite relations to the cerebrospinal fluid.

The cerebrospinal fluid.—The choroid plexuses of the cerebral ventricles are generally considered as the chief source of the cerebrospinal fluid. The evidence in favor of this concept is excellent, being based on many and varied observations.

The first hypothesis regarding the origin of the cerebrospinal fluid may be referred to Haller (1757), who considered it to be the product of the leptomeninges. Faivre, in 1853, and Luschka, in 1855, independently suggested the choroid plexuses as the elaborators of the fluid. The evidence for this view was histological, the glandular characters of the plexus causing fairly wide acceptance of the idea. Pathologically, also, the intraventricular origin of the fluid seemed established in those cases of hydrocephalus subsequent upon obstruction within the aqueduct or other interventricular channel.

More definite proof of the production of the cerebrospinal fluid by the choroid plexuses was afforded by observations made about 1900, in which pharmacological agents caused changes in the rate of flow of the fluid from cannulæ, the process being associated with histological changes in the cells of the plexus. Following these important observations many other workers have added valuable histological data in support of this viewpoint. This conception has been greatly strengthened recently by embryological findings which related the initial extraventricular flow of the fluid to the primary tufting of the ependymal cells to form the rhombic choroid plexuses. From these data—histological, pharmacological, and physiological—the function of the cells of the choroid plexuses as the elaborators of a characteristic body fluid seems established. It must be understood, however, that those structures, while undoubtedly producing by far the greatest portion of the cerebrospinal fluid, constitute merely the intraventricular mechanism for fluid-elaboration. There occurs also a further production of the fluid by the nervous system itself, an addendum carried by the perivascular channels into the subarachnoid spaces. Furthermore a minimal production of fluid by ependymal cells, negligible in its significance and total amount, may occur.

The cerebrospinal fluid elaborated by the choroid plexuses is poured into the cerebral ventricles, lined by smooth ependyma. That portion of the fluid formed in the lateral ventricles escapes into the third ventricle and thence by the aqueduct of Sylvius into the fourth ventricle. From the fourth ventricle the fluid is poured into the subarachnoid spaces; there is no good evidence that functional communications between cerebral ventricles and the subarachnoidal spaces exist in any other region.

Some uncertainty as to the exact mode of escape of the cerebrospinal fluid from the fourth ventricle into the subarachnoid spaces still exists. The weight of evidence surely inclines to the consideration of the *foramen of Magendie*—a median perforation of the inferior velum—as a true functional opening. This foramen has been termed by many workers an artifact, but embryological studies indicate that there is a true anatomical break in the membrane of the velum, a perforation of both ependymal and pial elements. The two lateral openings—*foramina* of *Luschka*—connecting the lateral recesses of the fourth ventricle with the subarachnoid spaces, seem to have as actual an existence as the medial opening. It is through these foramina, or surely in the region of the inferior tela choroidea if through an anatomically intact yet physiologically permeable membrane, that the cerebrospinal fluid produced in the cerebral ventricles passes into the subarachnoid spaces.

The cerebrospinal fluid, flowing through the roof of the fourth ventricle, enters the subarachnoid spaces in the medial cerebellar-bulbar angle. The immediate reservoir into which the fluid is poured is the cisterna cerebello-medullaris; from this region the further distribution of the cerebrospinal fluid is accomplished. This new product of the choroid plexuses slowly diffuses downward into the spinal subarachnoid spaces, but passes more rapidly upward around the base of the brain, and thence, more slowly, over the hemispheres, covering the whole cerebrospinal axis in a perimedullary arrangement.

The cerebrospinal fluid circulates through the subarachnoid spaces, which are efficient fluid-retaining channels. Evidence concerning the relative impermeability of the arachnoid membrane and of the pia mater is afforded by the spread of injections of various kinds. Such suspensions of granules are, of course, abnormal in every way, but are of importance in consideration of the power of cells to engulf bacteria and particulate matter; reliable tests of leptomeningeal permeability, however, may be made only with true solutions. In general, the arachnoid channels may be considered as fluid-retainers with unquestionable powers of diffusion or absorption in regard to certain elements in the normal cerebro-spinal fluid, deriving in this way a cellular nutrition.

In the subarachnoid spaces the cerebro-spinal fluid receives a small addition from the perivascular spaces, as briefly mentioned above. This is apparently derived from the "tissue fluids" of the nervous system, collected into perineuronal (pericellular) spaces, draining into pericapillary and perivascular channels. These pathways probably represent the accessory drainage of the nervous system, and pour forth a fluid somewhat different from the product of choroid

plexuses. The evidence for this statement concerns quantitative chemical differences between the ventricular fluid (product of the choroid plexuses) and the subarachnoid fluid (product of the choroid plexuses plus the perivascular system); the ventricular fluid contains more dextrose and less protein than does the subarachnoid.

The *drainage* or return of *cerebrospinal fluid* to the blood stream takes place from the subarachnoid spaces. It is a direct process, the fluid passing through no intermediate system, as is shown by the almost immediate appearance of soluble chemical bodies in the blood-stream after subarachnoid introduction.

There have been many theories proposed regarding the *absorption of the cerebrospinal fluid*, the first important one being that advanced by Key and Retzius in 1876. These observers considered the fluid to be absorbed through Pacchionian granulations (the hypertrophic arachnoid villi of adults) for the most part, with a lesser drainage into the lymphatic system. This view was generally accepted for a time, but with the realization that the Pacchionian granulations, as such, do not exist in infants and in the lower animals, it was felt that this view was inadequate. Following the anatomical work of Key and Retzius, many physiological experiments were attempted, demonstrating very conclusively the greater importance of the venous drainage. These observations dealt with the subarachnoid injections of true solutions (potassium ferrocyanide, methylene blue, fuchsin, phenolsulphonaphthalein, etc.), of colloidal suspensions (trypan blue), and of granular suspensions (cinnabar, carbon, etc.). From these varied methods of investigation, it has become quite well established that the chief drainage of cerebrospinal fluid takes place within the cranium and that absorption from the spinal subarachnoid space is much slower and much less in amount. The escape of fluid is largely into the blood-stream directly, the rate depending to a considerable degree upon the pressure of the fluid, suggesting a mechanism of filtration, though none of the experiments rule out the factors of diffusion and osmosis. More recent studies have shown that the rate of absorption varies directly with the size of the molecule of the foreign substance. No absorption of true particulate matter (even with particles as small as those in india ink) occurs to any appreciable extent, but a free and rapid absorption of all true solutions invariably takes place. Between these two types there have been found slower rates of absorption for colloidal solutions, the larger molecules being absorbed more slowly than the smaller.

As to the exact method of absorption of the cerebro-spinal fluid, several hypotheses have been advanced since Key and Retzius published their monograph on the nervous system. Most of the theories possess obvious limitations, such as the idea of a valvular mechanism leading into the great venous sinuses, or the idea of a

total lymphatic absorption. The evidence seems against the conception of a diffuse absorption of the fluid by either the cerebral or the meningeal capillaries, the latter being wholly untenable anatomically. Recent observations have shown that the chief channels for absorption are through the arachnoid villi into the great dural sinuses. These structures have been already described; the cerebrospinal fluid circulating in the subarachnoid spaces enters the villus and comes directly in contact with a double membrane of arachnoid mesothelium and vascular endothelium. Through this cellular layer free passage of fluid apparently takes place. Such a viewpoint has much in its favor; the methods of investigation leading to this conception were a combination of anatomical and physiological measures. The idea affords an anatomical basis for the physiological observation that the major absorption of fluid is cranial and not spinal; it is in keeping with all other phenomena observed in regard to the fluid.

Assuming, then, that the chief absorption of the cerebrospinal fluid takes place into the great venous sinuses of the dura through the arachnoid villi, there must be considered the much slower, much less efficient *drainage of fluid through the lymphatic system*, the fluid apparently escaping outward in potential spaces about each emergent nerve. From these perineural sheaths, drainage into the lymphatic system occurs. This is always outside of the dural cuff around each nerve, for the lymphatic system is not continued within the membranes of the central nervous system; connections between the cerebrospinal fluid and true lymphatic vessels are always indirect. This is of importance in the possible pathway of infection in cases of meningitis or poliomyelitis; the process may extend directly into the fluid when once the perineural space about a cranial nerve (as in the case of an olfactory) is entered.

Normally about 125 to 150 c. c. of cerebrospinal fluid exists within and surrounding the central nervous system. It is not known definitely what *amount of cerebrospinal fluid* is produced in man. The evidence surely points to a very active process of fluid elaboration and absorption. Most of the observations concerning the amount deal with the collection of the fluid from subjects with spontaneous or artificial cerebrospinal fistulæ. But in all of these cases, the pressure conditions are abnormal; the fluid is produced against only the resistance to flow in the fistula, instead of against the normal intracranial tension. Hence the amount collected is probably much more than is normally formed. The whole question of the relation of the cerebrospinal fluid (subarachnoid) pressure to vascular pressures is of interest here. Pressures in the great dural sinuses are practically the same as in the subarachnoid spaces. Perforations of

the sinus wall without release of fluid in the subarachnoid space, do not cause bleeding outward; such perforations may remain patent, suggesting a constant flow of cerebro-spinal fluid into the venous sinus. The subarachnoid and venous pressures within the cranium must, in consequence, be conisdered practically equal; the subarachnoid tension may be said to be constantly lowered to the venous pressure.

The cerebro-spinal fluid, then, circulates within the medullary canal systems and in the subarachnoid spaces surrounding the central nervous system. It may be considered a fluid-cushion, possibly protective in nature. But it has other more important functions—the nutrition of the avascular arachnoidea, the affording of channels of escape of perivascular fluids, and the maintenance of pressure-balances within the closed cerebrospinal system. Halliburton has recently likened the fluid to an ideal saline solution—physiologically balanced—a concept of great importance.

Clinical importance of the cerebrospinal fluid.—The spinal fluid, fortunately for the serologist, is always normally water-clear, practically cell-free, and contains only traces of salt and dextrose. As such it makes an ideal solvent or vehicle for transmission of the products of all manner of diseases which may lie on the banks of this fluid stream. Only sterile distilled water could be better. By drawing off a sample and subjecting it to examination we may learn much as to the territory it has drained. Although variations have been shown to exist in the fluid in different loci of the nervous system, these variations are not great when free communication exists between the several portions. However, this effect of inflammatory or traumatic agencies occasionally leads to pocketing of the fluid in one or another part, as in internal hydrocephalus caused by obstruction of the outflow of ventricular fluid by blocking of the fourth ventricle exit as result of meningeal inflammation or tumor. It is, then, fair to assume that, except in unusual cases, the spinal fluid represents an admixture from cerebral sources and that pathological changes which contaminate it in widely separated areas of brain and spinal cord will be recorded in the fluid drawn by lumbar puncture.

Technic of lumbar puncture.—Lumbar puncture, as its name implies, consists in puncture of the subarachnoid space in the lumbar region. We are limited in choice of site for puncture to the first, second, third, and fourth lumbar interspaces, for the reason that the cord extends in adults to the first lumbar vertebra, and below the fourth, mechanical difficulties prevent the puncture. As a matter of experience it is found that the second and third lumbar spaces are easiest and safest for the purpose, and the best landmark consists of a line drawn from the crests of the ilia with puncture just above or below this line.

The patient should be lying on his side but near the edge of the bed and with back well bent outward by dropping the head and flexing the thighs on the abdomen. The site of injection is then found as described, and 2 or 3 c. c. of cocain or novocain solution injected into the skin and subjacent tissue, 1 cm. outside of the mid-line and between the spines. With thumb held firmly on this inter-spinous space the needle with stylet in place is then carefully pushed inwards and slightly upward until a sudden giving-way, which is usually readily felt, assures the operator that the dura has been passed; then the stylet is removed. The distance is variable, but is seldom less than 4 cm. or more than 6 cm.

If difficulty is met it is best to draw the needle well out nearly to the skin and try a slightly different direction, or it may be best to insert the needle directly in the mid-line, the course of choice with many. If the needle still refuses to enter, experience teaches that it is advisable to use the interspace above or below, rather than persist at the same site. Occasionally the needle seems to the operator well pushed home and no fluid comes; this is usually due to an abnormally low spinal fluid pressure, which may be overcome by placing the patient in the erect position, when fluid will flow. The report of a negative puncture is almost never acceptable, being allowed only in abnormalities of the spine preventing the entrance into the canal or to filling of the dural sac with abnormal tissue, as in certain cases of tumor.

As to the choice of a needle most are now agreed. The essential features are (1) that it should be long enough, but not so long as to be awkward; (2) that the stylet reach exactly to the tip presenting a flat surface running up to the "point" which is more of an edge than a point, and (3) that it may be bent without breaking. For this last reason the nickeloid needle is greatly to be preferred to the steel one, the cost of platinum now being almost prohibitive.

It is easier to puncture with a small-bored needle than a large one, both for operator and patient, so that except in cases where pus is to be expected, a needle not greater than 1 mm. (No. 18) in diameter outside measurement will serve.

Contra-indication and after-effects of lumbar puncture.—There are two conditions in which puncture should not be done, or should be performed with great caution: (1) Where subtentorial pressure is excessive, withdrawal of fluid below has a number of times led to respiratory paralysis, due to jamming of the medulla in the foramen magnum. (2) It has been pointed out that in recent septic cortical wounds, withdrawal of fluid has separated newly-formed adhesions and caused dissemination of a focal meningitis. The after-effects of puncture are nil if the patient remains in bed. Headache, nausea, and vomiting may follow lumbar puncture in ambulatory patients—troublesome but never dangerous symptoms.

Tests.—Six c.c. of fluid are ample for all usual tests. Those which are likely to be of value may be grouped under five headings as follows: (1) Physical characteristics and naked eye appearance; (2) chemical; (3) histological; (4) bacteriologic; (5) biologic. Let us consider them in turn, laying especial emphasis upon the simpler tests of proved value which may be utilized promptly by the clinician.

Physical characteristics and gross appearance.—The *pressure* of the spinal fluid should always be taken. It may be determined roughly, with almost no loss of time, by allowing the fluid to run into a calibrated capillary tube, or with a mercury manometer. Neither of these methods is exact as understood by physiologists, but both are of sufficient clinical accuracy. It will be noted with the aqueous manometer (which has the greater range) that there are oscillations of a few millimeters synchronous with the pulse and that there are also variations of a centimeter or more with respiration. These respiration oscillations may be augmented by deeper breathing; but especially may the pressure be increased by holding the breath and by sudden respiratory movement such as coughing. It is largely because of these cardio-respiratory phenomena that such large variations in the normal have been given. However, in the adult patient with normal blood pressure, lying on the side and relaxed, the average pressure by mercury will be 5 to 7 mm., and over 15 will be a distinctly high pressure. The writer, using the aqueous reading, places normal about 150 mm.; increase of blood pressure may cause an elevation of fluid pressure to 250 mm., but a pressure of over 300 mm. must be explained on the basis of increased intracranial pressure. Brain tumors with choked disc of 3–4 diopters usually give pressure of 400 mm. and more.

Sub-normal or zero pressure are less well understood; in long-standing degenerated cord diseases we usually find a low pressure, and fainting promptly causes a drop in the fluid pressure to zero; and the pressure will be low if from any cause the fluid is prevented from passing down the spinal canal. Estimations of pressure in cerebral trauma should yield valuable information, especially in differentiating between acute cerebral edema and hemorrhage on the one hand and shock on the other. It has been indicated above that blood pressure and spinal fluid pressure are correlated, and this is to a certain extent true. Therefore, any marked variation in this relation is likely to be significant of intracranial mischief.

Color.—Normally water-clear; any variation must be considered abnormal. The fluid may be opalescent from suspended bacteria or cells; it may be slightly pink or yellow or yellow-green from the presence of altered blood-pigment; it may contain an obvious sediment of white, yellow, green or some color due to bacterial growth. The so-called "xanthochromic" fluid is lemon-yellow to deep orange in color (see below).

Bloody fluid must be distinguished from fluid contaminated with blood at the time of puncture. The skillful operator will almost never contaminate his specimen with blood in sufficient amount to be visible to the naked eye. If bloody fluid is obtained, at least three test tubes should be used and labeled in the order of withdrawal; if all are seen to contain the same amount of blood, and if after standing or centrifugalizing the supernatant fluid is yellow-tinted, the blood surely came from the central nervous system. Fluid contaminated with blood will show a diminishing blood content as more is withdrawn, and the supernatant fluid will be clear and colorless.

Clotting occurs in many fluids showing high proteid content; it is therefore to be expected in most cases of meningitis. In cases where the cord is excessively or rapidly compressed there usually occurs a fluid which clots with extreme rapidity; in fact, at times if the flow be sluggish, clotting will take place in the needle. Not infrequently the clot will in 10 minutes be so firm that the tube may be inverted without the loss of fluid. Owing to this characteristic and to its yellow color this fluid is usually spoken of as showing the "*syndrome de coagulation massive et de xanthochromie*" (of Froin), and as it is of great diagnostic importance in constricting lesions of the cord, a few words as to its probable formation will not be out of place. According to A. Mestrezat, owing to compression of the spinal cord, whether the agent be intra-medullary, meningeal, or extra-dural, and regardless of its pathological nature, the spinal canal below this point is separated from that above; the pressure of the fluid in this closed-off sac is lowered because spinal fluid pressure is dependent upon that of the cerebral fluid; into this stagnant sac transude albuminous substances together with pigment from broken-down blood, derived from interference with circulation at and near the point of spinal cord pressure. From this it will be seen that the excessive proteid content of the fluid is the essential element in the syndrome, rather than xanthochromia, which latter is found from hemorrhagic decomposition in any part of the central nervous system.

In tubercular meningitis a more or less characteristic clot is obtained, for, on standing for a few hours, a filmy clot is found stretching down from the surface of the fluid.

Chemical tests.—Tests for proteids and for sugar are the two which have been shown to be of essential importance.

Proteid is a normal constituent in the cerebrospinal fluid, but only in very small amount. It is increased as a result of meningeal inflammations of all kinds and as the result of transudations. In the former class we see slight increase only in chronic inflammatory conditions, such as tabes, rather more in general paresis and tuberculosis, and much more in the various acute meningitides. Supposedly as a result of transudation we frequently find moderate increase in cases

of vascular hypertension and brain tumor and greater amounts with cerebral thrombosis and following cerebral hemorrhage, while in cord compression we obtain massive readings. Proteid increase is called out in response to a great variety of diseases and disturbances of the central nervous system and its vessels; an increase should be looked upon as an undifferentiated response to the agency at work and in no way specifically diagnostic.

Decrease in proteid content occurs as a result of increased production of cerebro-spinal fluid; thus it is diminished in so-called "serous meningitis."

There are many tests for proteids, but unfortunately there is no simple reliable quantitative one, so that all depend upon an ocular determination of the amount of precipitate. The following are satisfactory:

(1) Add 95 per cent alcohol—three parts to spinal fluid one part. Normally a just perceptible white cloud indicates the precipitated proteids. Any precipitate more than this is abnormal.

(2) *Ross-Jones modification of Nonne Phase I test.*—Underlay a small amount of spinal fluid (0.5 c. c. is sufficient) with saturated ammonium sulphate; a white ring at the junction of the fluids developing in ten minutes represents proteid increase, presumably globulin. Normally there is no precipitate.

These two tests are perhaps the simplest and most time-saving of all. Other tests which have strong adherents are the Noguchi butyric acid, trichloracetic, and Pandy (carbolic acid) tests.

Sugar is also a normal constituent of spinal fluid. The importance of its determination is found in the meningitides where sugar is either absent or greatly diminished, the theory being that sugar is utilized in the growth of the bacteria.

Fehling's test.—Use solutions A and B 1/10 strength, add equal parts of mixed reagents and spinal fluid, and boil. A red color denotes sugar.

The *Goldsol reaction* is too complicated, because of the preparation of the reagent, for use except in a well-equipped laboratory under the direction of an expert, and hence the technic is not here given. It may be stated that the one great value of the Goldsol reaction lies in the diagnosis of general paresis as differentiated from other forms of nerve syphilis and brain disease which may simulate paresis. The "paretic curve" here obtained is unquestionably of value, although not pathognomonic, as frequently claimed. The "syphilitic curve" and the "meningitic curve" are of distinctly less importance and reliability. In this test blood-free fluid must be used, so that in traumatic cases of the central nervous system the applicability of this test will be greatly diminished.

Histologic (Cytology).—First among the methods of examination of the cerebrospinal fluid the study of its *cell-content* or "cytology"

has fallen into unmerited disrepute or, to put it less strongly, has simply been "omitted" in many cases. The presence of a leucocytosis affords ocular evidence of inflammation which no other test can refute.

Normally there are no cells in the spinal fluid, although a few may be found—usually large mononuclear cells presumably from the lining wall of the fluid spaces—which may be discounted. Inflammatory cells, lymphocytes, or polynuclear leucocytes have no place in a normal cerebrospinal fluid (although some admit an occasional lymphocyte as normal).

Cells had best be studied in one of two ways: (1) If many are present, as in acute meningitis, by means of stained smears; (2) if the fluid is clear or very slightly turbid, by means of the counting chamber.

(1) Smears are prepared in the usual manner for studying blood, and best stained with Wright's blood stain or methylene blue and studied under high power.

(2) For studying the cells of a clear fluid the counting chamber method is recommended because (*a*) it is difficult to get a small number of cells onto a slide and more difficult to keep them there during staining, and (*b*) the number per cu. mm. has considerable value in such clear fluids. Either acetic acid 5 per cent or a stain (methyl violet 0.2, acetic acid 2.0, water 50.0) is drawn up to mark 1 on the counter, and then cerebrospinal fluid to 11. After shaking for two minutes the counting chamber is filled and the count made, the process is repeated, an average struck, and calculation made to read for the number of cells per 1 cu. mm.

Several points must be touched upon. A count should be made under high power, for two reasons: In order to be certain that cells are counted, for there is apt to be confusing débris in cerebrospinal fluid, and, secondly, that a rough differential count may be made. The cell-count should be made soon after withdrawal of the fluid before there has been any clotting, if the count is to be of value. This troublesome necessity, however, may be overcome by making the dilution, shaking and sealing the ends with a rubber band or a Seymour protector. It is of advantage for convenience and accuracy, though not necessary, to use a Fuchs-Rosenthal counting chamber or Zappert-Ewing ruling so that more cells may be counted.

What cells do we find? Chiefly three varieties:

(1) The polynuclear leucocyte, denoting acute infection.

(2) The lymphocyte, denoting chronic inflammation and acute poliomyelitis, chiefly.

(3) The large mononuclear cell, which, as has already been said, may be found normally and which appears in excess in all inflammatory states.

We also recognize plasma cells and phagocytic mononuclear cells, and, of course, red blood corpuscles if not destroyed by the diluent. Rarely tumor cells have been reported.

The cell-study not only gives us information of value for diagnosis, but for prognosis also; for instance, a change from a polynuclear to a lymphocytic formula together with an increase in number of phagocytes is indicative of the subsidence of an acute meningeal inflammation.

Bacteriologic.—Smears are made from pus in the usual way and usually are studied in connection with the cytology. In acute infections—especially where meningeal symptoms are present—it is wise to make cultures, even if the fluid be clear, for in the earliest stages of meningitis there may be no ocular abnormality. Glucose agar is satisfactory for the growth of most forms of bacteria likely to be met with, though the addition of a little fresh blood makes a more favorable medium for the meningococcus. It is wise also to incubate a tube of the spinal fluid itself, either for the growth of bacteria or as a source of supply later should such be necessary. For the analysis of cultures the reader is referred to works on bacteriology. As a matter of experience the organisms met with most frequently in civil practice are the meningococcus, streptococcus, and staphyclococcus, more rarely B. typhosus, B. influenzæ, and the pneumococcus.

Unfortunately we are unable as yet to recognize the organism of poliomyelitis, or to recover the spirocheta pallida by culture. In the latter case fluid from very active syphilitic meningeal cases will at times yield the organism when injected into the testicle of a rabbit, but this is of academic rather than practical interest.

The tubercle bacillus should be found—usually after prolonged search—in fluids from tuberculous meningitis. Great care should be exercised in preparing the film. For this, take the web-like clot which always occurs in such fluids, carefully ensnare it in the platinum loop, and transfer to as small an area on the slide as is possible, then stain in the usual manner. If not found, inoculation into the peritoneal cavity of a guinea pig may be practiced. But this again is hardly a routine procedure, in that the time required for the development of tuberculosis is four to six weeks. Methods to hasten this period of incubation are not reliable.

Biologic.—Under this heading comes the Wassermann reaction. The performance of this, like the Goldsol test, is a matter for the expert laboratory worker and need hardly concern us here. A few words may be said, however, as to its clinical application. Like all laboratory evidence, this must be most carefully correlated with the clinical picture presented; the tendency is for some to overrate its

significance, for others to underrate it. A positive Wassermann reaction in the spinal fluid means that the patient *has or has had* syphilis of the cerebrospinal axis, but it does not necessarily mean that a given lesion is syphilitic. On the other hand, a negative Wassermann does not signify that the patient may not now be suffering from the effects of previously active syphilis, though in the case of a young man this would be most unlikely. Again, it is seldom wise to rely upon either cerebrospinal or blood Wassermann alone. Both should be examined. Especially important is it that the reactions in a suspected case should be carefully correlated with an adequate neurological examination.

Summary of tests.—As the cerebrospinal fluid is examined for different purposes we naturally can not use a hard-and-fast scheme. The following should give the greatest amount of useful information with the least waste of time: In every case note pressure, color, turbidity, proteids, and cells. If normal or bloody, no more need be done, as a rule. If there is evidence of acute meningitis, carry out bacteriologic work. If there is evidence of chronic meningitis, examine especially for tuberculosis and syphilis.

In the summary below an attempt is made to give the characteristic spinal fluid picture as found in a number of disease entities and symptom groups. In order to simplify this table, qualification has for the most part been eliminated, so that there will be found many purposeful omissions.

Increase of intracranial pressure, from all sources	The fluid will be under increased pressure provided that free connection exists between cranial and spinal subarachnoid spaces.
Tumors	Frequently a slight increase of proteids, but without cells.
Hemorrhage connecting with subarachnoid space	Free blood present in all tubes.
Hemorrhage into deep tissue	Yellow tinted fluid with moderate increase of proteid, frequently increasing in amount for several days.
Sinus thrombosis	Yellow tinted fluid with marked increase of proteid and usually some red cells. Also usually leucocytosis.
Arterial thrombosis	Usually slight increase of proteids.
Meningitis, acute	Fluid almost always turbid from cells and bacteria—may be colorless, yellow, green, or brown. Microscopically shows polynucleosis and usually the organism concerned. Cultures are usually successful, but special media may be required.

Subacute stage	The fluid begins to clear, the cytologic formula changes from polynuclear to lymphocytic and phagocytes are seen. The organisms disappear. Tests for proteid are last to improve.
Tubercular meningitis	The diagnosis must frequently be based upon a clear or slightly turbid fluid showing a moderate lymphocytosis, with a considerable number of polynuc. leucocytes, and upon the web-like clot. T. B. should be sought for and often are found. Inoculation into a guinea pig may be required.
Poliomyelitis	Is suspected when a fluid with moderate lymphocytosis and moderate proteid excess is found. It is, of course, not diagnostic.
Chronic stage	Findings show considerable variation, not only between different cases, but at different times in the same case. Usually a high protein content with moderate lymphocytosis, the intensity of reaction fluctuating with the walling-off or canalization of the subarachnoid space. Pressure readings are here useful in determining interruption to flow, at times adhesions entirely preventing fluid from reaching the lumbar sac.
Syphilis of the central nervous system	The fluid picture varies with the activity of the syphilitic process and with the extent and virulence of the meningeal involvement. In general, the "active" case gives a moderate lymphocytosis with increase of proteid (notably globulin) and a positive Wassermann. The "inactive" or "degenerative" case gives similar reactions, but much less intense. Goldsol curves are of value in differentiating types of neurological syphilis.
Cord compression from all sources	1. Rapid and marked compression gives the following picture, characteristic, if not pathognomonic: a yellow (so-called xanthochromic) clear fluid, obtained under low pressure, which clots spontaneously and rapidly, usually cell-free or nearly so, but showing massive protein precipitation. 2. Slow and incomplete compression gives a colorless fluid, but with marked excess of protein, without, however, signs of exudation.

Inflammatory processes in the meninges.—Inflammation of the dura is called pachymeningitis, and of the inner coats (arachnoid and pia mater), leptomeningitis. With the exception of localized inflammations and those following injury, inflammation of the meninges nearly always involves the coverings of both the brain and spinal cord.

Cerebral pachymeningitis.—This may affect either the outer or inner surfaces of the dura, occasioning an external or internal pachymeningitis. *External pachymeningitis* nearly always results from injury to the skull, and is not as common as is usually thought. It may be secondary to disease of the overlying bone, especially in syphilitic, tubercular, and carcinomatous conditions.

Internal pachymeningitis is rare. It is sometimes hemorrhagic in nature, a pathologic condition characterized by repeated extravasations of blood on the inner side of the dura and associated with the development of large capillaries. This usually occurs in old persons, especially in those who are either arteriosclerotic or alcoholic. It is frequently found in some forms of insanity. It may be present in conjunction with external pachymeningitis, especially in purulent, syphilitic, and tubercular inflammations.

Symptoms.—The symptoms of pachymeningitis, whether external or internal, are definite, and depend upon the pressure exerted on the brain. Generally the patient complains of headache, and rarely of tenderness localized to the inflammatory area, the specific symptoms depending upon the part of the brain involved. If in the motor area, there will be irritative symptoms such as Jacksonian convulsions, which may be followed by a greater or lesser paralysis; if over Broca's area, motor aphasia; if over the parietal areas, where the pachymeningitis is most common, there may be irritative pains or paresthesia on the other side of the body, accompanied sometimes by disturbance of sensation; if over the temporal lobes, sensory aphasia; if over the occipital convolutions, disturbance of vision on the other side. There may sometimes be loss of consciousness, delirium, or stupor, or there may be no symptoms at all.

Summary.—History of a preceding injury to the head or of alcoholism, or syphilis, or the presence of symptoms indicative of tumor of the bones of the skull. The symptoms may come on rapidly or slowly, and consist of diffuse headache, possibly unconsciousness, coma or delirium, with irritative phenomena, such as convulsions on the other side, paralysis of various kinds, either partial or complete motor or sensory aphasia; or, what is important, there may be no symptoms at all. It is evident that the diagnosis of a pachymeningitis is very complex and depends upon the location and character of the inflammation.

Spinal pachymeningitis.—Isolated inflammation of the spinal dura without involvement of the membranes underneath is very unusual and its occurrence is doubtful. In some instances, however, the dura is preponderantly involved. As a rule, inflammations of the dura are secondary to disease of the vertebra, as in tuberculous, syphilitic, carcinomatous, or sarcomatous inflammations. It is possible to have a syphilitic pachymeningitis without involvement of the vertebra, but in most of these cases the pia and spinal cord are also diseased. The symptoms of a spinal pachymeningitis secondary to vertebral inflammations have already been discussed under the latter heading.

Cerebrospinal meningitis.—In most cases the leptomeninges of the brain and spinal cord are involved at the same time, and it is only rarely that either is involved alone. Inflammations may be of various kinds: epidemic, purulent, tuberculous, and serous. Syphilitic meningitis has been discussed under the head of syphilis.

Leptomeningitis.—Acute inflammation involving the pia-arachnoid may occur through the entrance of the irritating or infecting agent by way of the blood-stream or through direct introduction of the infection from without. The exudate in the latter types of cases may be localized largely within the subdural space. This is rare and is seen only in the cases of perforation of an abscess into the cavity. In others and most of the cases of direct infection the exudate is seen also and in large part in the subarachnoid space. In the acute inflammatory types which gain entrance by way of the blood, the inflammation is usually very diffusely distributed over the whole surface of the brain and cord. In other cases, and in many of those which result from rupture of abscesses, it has a basilar localization.

Meningitis may occur as a primary infectious disease or it may occur secondarily in the course of many infectious processes. *Infectious cerebrospinal fever* is a disease due to the diplococcus intracellularis (Weichselbaum). It occurs in epidemics or may occur apparently sporadically in isolated neighborhoods. The organisms are found in secretions of the nose and mouth of patients and often in otherwise healthy individuals. Abortive cases are not uncommon. It is widely circulated and is most likely to become a plague when large numbers of persons are crowded together. The disease is commonly seen in infants, although persons of any age may be attacked. Army camps are very prone to such epidemics.

In this disease the brain and cord are inflamed throughout, although there are forms in which the inflammation is intense around the base of the brain, whence it may extend into the ventricle, leading to an obstructive hydrocephalus. In many cases the inflammation is more intense elsewhere. The exudate is rich in polymor-

phonuclear cells and is largely confined within the subarachnoid space which becomes distended with pus. The pus is often very thick and can not be easily removed by lumbar puncture.

The symptoms vary in the different cases. The onset may be very sudden, with headache, rigor, spasms, coma, and death in 24 to 48 hours. In the ordinary cases the disease is ushered in by chills, headache, and vomiting. There are pains in the back and extremities, restlessness and often spasmodic movements. The cranial nerves may become paralyzed. Herpes and stiffness of the neck are frequently present. In the basilar form the head is retracted. Kernig's sign is usually positive. The pulse is rapid and irregular. The fever is low but irregular. It often becomes high at the end. The respiration may be disturbed, and toward the end the Cheyne-Stokes variety has been seen. There is a marked leucocytosis.

A large majority of the untreated cases die within the first five days. Death may be delayed, however, for a very long time, resulting finally from complications.

Complications are numerous. Internal hydrocephalus in the basilar forms, epilepsy from adhesions, paralysis from cerebral thrombosis, persisting encephalitis, etc., are noted.

The diagnosis is readily made clinically and easily confirmed by lumbar puncture.

The treatment of acute cerebrospinal meningitis is both symptomatic and specific. The specific treatment concerns repeated lumbar punctures and the introduction of specific serum—that of Flexner. The serum is introduced after the removal of the cerebrospinal fluid. The quantity introduced is governed largely by the amount of fluid withdrawn. During the earlier severe periods, lumbar puncture should be made every eight hours or even more frequently. After the first or second day less frequent treatments are usually advisable.

The frequency of the treatments can be determined often by the cell count and bacterial count of the exudate. This is not always true, however, when the pus is very thick. Cases are seen frequently where the pus is so thick that it will not flow through the needle.

In the basilar forms ependymitis and hydrocephalus are frequently present. Ventricular puncture is indicated, and the introduction of serum into the ventricle may be of value. These cases are very frequently fatal. Again in these cases, lumbar puncture should follow ventricular puncture. When puncturing the ventricle the fluid must be allowed to escape slowly; a rapid flow may lead to greater extension of the infection along the ependyma.

Meningitis follows acute infections.—Acute cerebrospinal meningitis is not as serious a malady and as difficult to treat as many of those cases of meningitis which follow such acute infections as pneu-

monia, influenza, etc., or those which result from perforation of abscesses of the skull, fractures, puncture wounds, or rupture of an infected sinus or phlebitic abscess. A great variety of different kinds of organisms may be seen in these cases; the pneumococcus, the streptococcus, the staphylococcus aureus, albus or citreus, the bacillus typhosus, bacillus coli communis, bacillus aërogenes, and others have been found.

As already mentioned, the pathological picture may vary widely. In the purulent forms, the pus may be located almost entirely in the subdural space. This is rare. The subarachnoid space is most frequently involved. In the case of perforation the pus is frequently localized. It may be only over the cerebrum or the cord or the part near the point of entrance. In cases of ruptured abscesses, sinuses, etc., it is frequently localized over the base of the brain and the cord. In these cases the lumbar fluid may be clear and sterile and give no aid in the diagnosis.

Again, especially in pneumonia, its distribution is uniform throughout the subarachnoid space. The pathological picture resembles acute epidemic cerebrospinal meningitis in every way except bacteriologically.

The cerebrospinal meninges, like the peritoneal cavity, may resist the introduction often of large numbers of bacteria. Although it is evident that mere introduction of bacteria of the ordinary variety may not cause meningitis, the other conditions leading to it are unknown, and the greatest care must be taken in operating and exposing these surfaces. Operating through infected areas in the scalp, bones, dura mater, although these infections may be very slight or almost healed, frequently leads to meningitis. Subsequent opening of the mastoid or other sinuses near a former trephine opening may be fatal. Through the loose tissue under the epicranium in such cases pus may readily gain access to the cerebral meninges.

These cases are generally difficult to treat. Lumbar puncture or ventricular puncture is an aid. Irrigations from cerebrum to lumbar region have been recommended. In cases of infections from puncture wounds, opening and free drainage are indicated. Specific treatment when available may be of assistance in some cases.

Tuberculous meningitis is common in children but may occur also in adults. In most cases it is a part of a general miliary tuberculosis. Again, however, the meninges alone may be involved. The infection reaches the meninges probably by way of the blood-stream from a primary focus in other parts of the body. This primary focus may be very small and is frequently overlooked even at the autopsy table.

In tuberculous meningitis the meninges of the base of the brain and of the spinal cord are frequently the chosen sites for the infection. In a few cases one may see the entire surface of the brain involved.

The lesions are generally in the form of small tubercles with central caseous matter, situated in the wall of blood vessels. All the neighboring portions of the subarachnoid space are filled with a fibrinous exudate which contains a small number of wandering cells, many of which show degenerative changes. The involvement of the subarachnoid of the base may lead to internal hydrocephalus, a most frequent occurrence in this disease.

The *clinical symptoms* are variable. The disease may have a very acute onset resembling the epidemic form. As a rule the onset is much less abrupt. Later the patient shows symptoms of internal hydrocephalus and increased cerebral tension associated often with transient paralysis. The temperature may be normal or subnormal. The spinal fluid is usually clear or slightly turbid. It contains an increase of small mononuclear cells, and a careful search usually reveals the presence of tubercle bacilli.

The treatment is symptomatic. Lumbar and ventricular puncture are of service.

Serous meningitis-meningism.—This is a form of meningitis in which there is an effusion into the meninges, but in which there is no true exudation comparable to that occurring in the purulent variety.

Under the term meningism, meningismus, or pseudomeningitis has been described that clinical variety in which the symptoms of meningitis are present, but in which pathologically and by lumbar puncture nothing is found perhaps beyond a congestion of the vessels and edema of the tissues. It is probably merely the primary stage of a serous meningitis. If the disease develops further into the second stage or stage of effusion, there will be what is commonly termed serous meningitis. Such meningeal changes, whether of an irritative or of an effusive nature, may be likened to similar pathologic conditions occurring in the internal organs, as in the various stages of a pleurisy or pericarditis.

Pathologically, in meningism there will be found a congestion of the blood-vessels with little or no edema, and rarely an organism causing the disease may be found in the meninges. In serous meningitis a somewhat similar condition holds, but in this case the serous effusion is associated with an increase of the lymphocyte elements. Only rarely will a specific organism be found.

Symptoms of meningism.—This may occur in the course of or follow any infectious disease, such as pneumonia, typhoid, rheumatism, scarlet fever, measles, or grippe. It is not difficult to recognize, for

there will be present those symptoms which are commonly termed meningeal, such as pain along the back or limbs, often of a numb character or at times sharp and shooting. The principal complaint is headache, especially in the occipital region. Besides, there will be rigidity of the head and back, and unwillingness to move the limbs for fear of increasing the pain. Sometimes there will also be hyperesthetic areas in different parts of the body. Rarely there may be muscular twitchings in the limbs and a general increase of the reflexes. Cerebrospinal fluid obtained by lumbar puncture is negative. The onset is generally acute. The temperature may or may not be increased, and the pulse and respiration are not much altered. The duration of the disease is usually short and the prognosis always favorable. Sometimes, however, there may be a complicating serous effusion.

Serous meningitis.—This may involve either the membranes of the brain or of the spinal cord alone, or both. There will be in addition to the symptoms enumerated above, which may occur first, pressure symptoms resulting from increased accumulation of fluid, their intensity depending upon the degree of the tension. When the spinal cord is principally involved, there will be in addition to the meningeal symptoms, pains in the limbs, girdle sensations around the waist, and, because of pressure upon anterior and posterior nerve roots, and later on the spinal cord, disturbance of sensation, increased reflexes which are later lost, and bladder and rectal phenomena. Lumbar puncture will always demonstrate an increase in the intraspinal tension. In most cases the disease lasts only a few weeks and terminates by complete recovery.

In the cerebrospinal forms, besides the symptoms enumerated, there will be some disturbance of consciousness and greater rigidity of the head, neck, and limbs, with occasional swelling of the optic nerve-heads. More rarely there may be temporary diplopia and disturbances in temperature, pulse and respiration. Lumbar puncture will, of course, demonstrate heightened intraspinal pressure, with increase in the amount of fluid. In most instances the symptoms will subside in a few weeks, the patient getting well. If, however, the disease continues, there will develop a serous effusion in the cerebral ventricles, with its accompanying symptoms of intracranial pressure.

Circumscribed serous meningitis may occur in the spinal cord and generally involves the lower portions, although it may be found in almost any part.

Cerebral serous effusion.—A serous effusion into the ventricles may be the beginning of a general serous meningitis, or may be confined only to them. The same causes which are active in the production of a serous meningitis may produce an internal hydrocephalus.

Pathologically there will always be found an internal and at times an external hydrocephalus, or an increase of fluid in the cortical meninges. Histologically there may be a cloudy swelling and proliferation of the ependyma, accumulation of cells under the ependyma, and cellular infiltration in the brain and spinal cord substance, and its meninges, especially along the blood vessels. The choroid plexus may be diseased; its over-action is supposed to be a possible cause of the increase of the fluid.

Circumscribed serous meningitis may occur in the pia-arachnoid of the cortex. Its symptoms will in no way differ from those of tumor of that part.

Hydrocephalus.—Limitations do not permit an extensive discussion here of the subject of *hydrocephalus*, but the relations of this pathological increase of fluid are of importance in the understanding of serous effusions within the nervous system and the meninges. The circulation of the cerebrospinal fluid has been divided into an intraventricular (ectodermal) course and a meningeal (mesodermal) course. It is of interest that obstructions to both divisions of the cerebrospinal pathways occur; blocking in either case leads to the development of an internal hydrocephalus. This type concerns the enlargement to varying degrees, of the cerebral ventricular system, particularly of the lateral ventricles where the opportunity for the dilatation is so much greater. As a general rule, the enlargement is symmetrical, although in certain intraventricular blocks, asymmetry may be pronounced. The augmentation of the size of the ventricles results in the increase in the intracranial tension with possible flattening of the cerebral convolutions, and, in extreme cases, the formation of endocranial markings.

It is quite easy to understand the dilatation of the ventricles (particularly of the lateral) in cases of intraventricular block. Excluding from consideration any possible congenital malformations, the obstruction to flow is practically always a mere mechanical cause of the hydrocephalus. The block usually occurs at the narrow places in the ventricular system as at the foramina of Monro, in the aqueduct, or in the region of the fourth ventricle. The common causes of such obstructions are cerebral tumors (including solitary tubercles and abscesses), which directly or indirectly cause obliteration or partial constriction of the fluid-pathway. The secretion of the cerebrospinal fluid by the choroid plexuses continues under such circumstances, and the ventricles suffer enlargement due to accumulation of fluid within them. There is practically no intraventricular absorption of cerebrospinal fluid; the production of fluid in such case continues until the intraventricular pressure becomes great enough to prohibit further production or until an abnormal balance between

production and absorption is reached. A unilateral obstruction of the foramen of Munro is the common cause of asymmetrical hydrocephalus. The enlargement in this variety of case is a purely mechanical process, due to local obstruction to the flow of fluid.

It is far more difficult to understand how obstruction to the flow of cerebrospinal fluid in the meninges results in the dilatation of the cerebral ventricles. Instead of an internal hydrocephalus with compressed hemispheres, one might expect an external type with thinned-out hemispheres and increased subarachnoid fluid. But the type of hydrocephalus resulting from meningeal involvement is invariably internal (enlargement of ventricles). Explanations for this phenomenon are not wholly satisfactory. The block within the subarachnoid space may occur in the cisterna cerebello-medullaris around the foramen of Magendie or it may be a more diffuse blocking of the leptomeningeal channels around the cerebrum resulting from the formation of inflammatory exudates and later fibrosis. It may be that the ultimate block in all cases occurs within the arachnoid villi, but of this there is yet no evidence. Suffice it to say that the obstruction here is meningeal and that it is practically always inflammatory (in acute or chronic infections of the cerebral meninges).

An interesting clinical differentiation between the two types of internal hydrocephalus may be made. In the intra-ventricular type (obstructive) no communication (or at best a very slight one) exists between cerebral ventricles and subarachnoid space; after lumbar puncture in such cases the fluid is quite rapidly replaced, indicating an extra-ventricular origin of at least part of the fluid. On the other hand, in the meningeal or communicating type, free connections between lumbar subarachnoid space and the cerebral ventricles exist.

The surgical measures aimed at the relief of internal hydrocephalus will not be discussed here, nor will the interesting occurrence of the condition in children with enlargement of the cranium be commented on. The problem of hydrocephalus concerns the alleviation of the increased intracranial tension, by temporary or permanent measures; the dilatation of the cerebral ventricles is a secondary mechanical sequel of the accumulation and increased tension of the cerebrospinal fluid.

Internal hydrocephalus resulting from serous effusion, as a rule, comes on in early childhood, and is not difficult to recognize if the process is active. Very often, however, there may be only mild symptoms such as have been described under meningism, only to have later in life either an acute or a chronic serous meningitis or internal hydrocephalus. In fact, many writers consider that serous meningitis or serous effusion in the ventricles in the adult is only an acute exacerbation of an old process which had its origin in childhood. However that may be, there is no question but that in the adult a

serous effusion may develop either acutely or gradually in the ventricles and cause symptoms which are usually recognized as occurring in brain tumor and from which it is sometimes almost impossible to make a differential diagnosis.

If internal hydrocephalus develops acutely, there will be, as a rule, an accompanying high fever, and the course of the disease will be rapid, terminating either in cure or death. Headache, nausea, vertigo, vomiting, and disturbance in vision and choked disc, sometimes of high caliber, are prominent symptoms. Besides, there may be paralysis of some of the cranial nerves, especially of the sixth, either of one or of both sides, and there may develop also cerebellar asynergy. Consciousness is nearly always clouded. The disease may last a week or two, terminating in quick recovery, leaving behind slight atrophy of the optic nerves, but no other symptoms. Sometimes there may be a recurrence of the disease, this terminating also in either recovery or death.

If, however, the symptoms of internal hydrocephalus come on gradually, the *differential diagnosis from brain tumor* will be very difficult. There will be present all of the pressure symptoms, such as headache, nausea, vomiting, vertigo, and choked disc, and because of the pressure exerted upon the motor fibers in the internal capsule there will result weakness and spasticity of the limbs, with increased reflexes and sometimes the Babinski reflex. There may also be paralysis of the external rectus, on either one or both sides. Because of the pressure on the cerebellum there will result incoordination in walking and sometimes incoordination of the eyeballs, or nystagmus. The differential diagnosis from cerebellar lesions is sometimes very difficult, but can be made principally on the fact that in cerebellar tumors there is hardly ever involvement of the limbs on both sides, and the asynergy is more acute and much more marked.

It must also be remembered that internal hydrocephalus may also accompany tumors of either the cerebrum or cerebellum, and in such case there will be, in addition to the symptoms resulting from the tumor, spastic paresis of the limbs with increased reflexes and the Babinski phenomenon. The prognosis in most cases of uncomplicated internal hydrocephalus is not very good, but sometimes complete recovery ensues, either as a result of operative interference, anti-specific treatment, or sometimes spontaneously, leaving behind nothing but a slight atrophy of the optic nerves.

Infection of the brain.—The general process of infection within the central nervous system differs somewhat from similar processes elsewhere in the body. The difference lies largely in the reactive phenomena of the tissues, and concerns anatomical and physiological

causes. It may be of advantage to discuss even briefly some of these interesting characteristics of the brain which so modify the spread of infective agents and so alter the general tissue reaction.

The general soft consistence of the brain is a gross characteristic which offers explanation for some of the peculiar reactions of the tissues to infection. The brain is largely made up of nerve cells and supporting elements. Of the latter, two great classes of tissues constitute the essential structure (excluding from consideration the ependymal elements)—neuroglia cells and white fibrous tissue. The epidermal neuroglia really forms the true support of the nervous tissues, occurring to a large extent in the gray matter and to a lesser extent in the white. The support that these glial cells add to the nervous system is apparently sufficient for the maintenance of normal cohesion of the tissues but does not afford any great excess of strength. On the other hand, the true fibrous tissue of mesodermal origin is related to the nervous system only as the supporting tissue of the meninges and as the fibrous walls of the invading blood vessels. This latter situation probably furnishes most of the connective tissue in the inflammatory processes within the substance of the brain, while the meningeal tissues add to the external support.

With such a conception of the supporting elements of the nervous system, it is easy to realize that the mechanical limitations to spread in other tissues are largely lacking here. Thus it is impossible to conceive of fiber tracts in the white matter of the nervous system as serving at all efficiently as barriers to the spread of infection as do fascial planes in other parts of the body. Certainly with the gray matter of the brain and also elsewhere in the nervous system there exist no anatomical structures which could modify in any way the passage of infection into it. The resistance here must be of a reactive character on the part of the fixed cells of the neighborhood and of blood elements brought to the focal point of infection. This lack of mechanical hindrance to the spread of septic agents is of significance.

The pathological processes occurring in infections of the brain are further modified by the fact that the fluid-drainage of the tissue is different from that in other parts of the body. True lymphatics are wholly lacking here, but an accessory drainage is afforded by the perivascular spaces. These channels drain outward from perineuronal or pericellular spaces through pericapillary and perivascular channels directly into the subarachnoid spaces. Thus a direct fluid-communication from brain tissue to subarachnoid space is afforded. This channel is important in affording a course for infection to travel from the meninges into the brain substance or from the brain substance outward.

The inelastic character of the dura modifies in considerable measure the course of infection within it. This is due to the fact that the intradural capacity is fixed and the space is entirely filled with three main elements—nervous tissue, blood, and cerebrospinal fluid. The capacity of this rigid system is fixed for the individual; all changes of volume of one or the other of the elements must be at the expense of the remaining. With the swelling incident to any inflammatory process following infection, certain readjustments of the three elements occur. In the initial stages of the infection (with the swelling confined to the brain) sufficient compensation is afforded by dislocations of the cerebrospinal fluid, probably in greater part into the blood-stream but with a further possibility of a change in the fluid content of the brain at the expense of the cerebrospinal fluid. A slight compression of the veins in the immediate neighborhood of the swelling likewise allows some early compensation. In subsequent stages, with an increase in the inflammatory swelling, a venous congestion ensues, with final cerebral anemia. In such inflammatory reactions with increase in brain-bulk, there is associated the obscure condition of cerebral edema, a watery swelling of the brain substance. This edema necessitates further dislocation of the cerebrospinal fluid and forces a further rearrangement of vascular conditions. Increases in intracranial pressure, however, are possible only up to physiological limits, and the compensatory mechanisms for the adjustment of intracranial or intradural volume must be effected with pressures under the physiological limit.

Such anatomical and physiological considerations render more intelligible the process of infection within the central nervous system. The infection of the brain tissue itself, "encephalitis," may be diffuse or it may be confined about an area of necrosis—a true brain abscess. Quite similarly the process of infection within the meninges may be strictly local or it may spread into a widely extending inflammatory reaction involving the entire extent of the cerebrospinal spaces. These different phases of infection within the nervous system will be considered here.

Diffuse encephalitis.—This term is customarily applied to the widespread inflammatory involvements of cerebral nervous tissues without any localized areas of necrosis or softening. In such cases, usually acute in nature, the inflammatory exudate occurs within the substance of the gray and white matter; it is characterized by extensive infiltrations of inflammatory cells, chiefly of the polymorphonuclear variety.

The etiology of these infective processes is not entirely clear. Perhaps one of the chief causes of such a widespread invasion of the nervous tissues is a direct infection from meningitis; the protective

mechanisms of the pia mater are overcome, permitting a generalized growth of bacteria. Against such an extension, the cellular reaction offers the only barrier. Under these circumstances, the infection is at first localized to the gray matter of the brain, but later extensions may carry it into the white.

Under the heading of diffuse encephalitis must be considered also similar conditions following some of the acute infectious diseases (typhoid, influenza, etc.). There have been described diffuse inflammations of the brain following trauma and also certain toxic conditions. Cerebral involvements following the acute infectious diseases are almost all hematogenous in origin, while the cases resulting from trauma must be considered either as direct infections or as those in which the trauma offered a place of lesser resistance for the lodgment of organisms. Actually the etiological factors in the diffuse inflammation are quite similar to those of brain abscesses.

Cushing gives the symptoms of an acute diffuse encephalitis to be those of all severe cerebrospinal affections, and considers them due to heightened intracranial tension—headache, somnolence, vomiting, fever, delirium, rapid pulse, etc. In the more severe cases, choked disc, coma, slow pulse, and changes in the respiratory mechanism may occur. Paralysis or irritative symptoms of an epileptiform character may occasionally be present.

Cerebral abscesses.—Localized infections of the brain tissue are usually brought about through any one of the following agencies: (1) by the blood stream; (2) by direct extension from a local focus of infection, and (3) by the introduction of septic material through perforating wounds.

(1) Under the heading of *hematogenous infections* must be classified the secondary abscesses or small areas of encephalitis met in any pyemia resulting from a focus of infection elsewhere in the body. Such metastatic cerebral abscesses may accompany an ulcerative endocarditis or a pulmonary gangrene, and, more rarely, typhoid fever, acute rheumatism, scarlatina, influenza, etc. In these cases the infection may be due to the specific bacterium producing the primary disease, but it is not infrequently brought about by secondary infection.

The likelihood of cerebral lodgment of infected emboli seems great, especially in those cases in which the organisms may have a particular virulence within the central nervous system. Bull's very interesting experiments with injection of massive doses of streptococci derived from poliomyelitic and nonpoliomyelitic patients show that the localization of the infective process within the nervous system is common. In a large series of inoculated animals, the two classes of streptococci gave the same high percentage of cerebral or meningeal involvement, 30 per cent. The pathological

findings within the central nervous system of these animals were largely focal areas of encephalitis, with typical infiltration of inflammatory cells and small areas of necrosis. In the membranes of the brain, typical acute leptomeningitis was produced, an extraordinary finding except when considered in connection with the frequent association of encephalitis, the whole process being a combined encephalo-meningitis.

(2) The problem of a *direct extension of an infection* from the outside into the cranial cavity is quite simple. The process usually starts as an infection of one or other of the air sinuses of the skull and extends by direct involvement of the intervening tissues into the central nervous system. These local infections are largely those of the frontal sinus (with resultant involvement of the frontal lobe), of the middle ear (with resulting involvement of the temporal lobe), and of the mastoid cells (with resulting involvement of the cerebellum or temporal lobe). In all cases the process is about the same: The involvement and subsequent necrosis of the intervening bone; the resistance offered to the spread of the infection by the dura; the more or less slow overcoming of this resistance with extension into the subdural space, but probably not until the inner surface of the dura had adhered to the outer surface of the arachnoid; the direct invasion of the arachnoid tissue and subarachnoid space with the gradual closing-off of this space; the slow involvement of the nervous tissue itself, with probable spread of the infecting agents into the brain substance along the perivascular channels; the final establishment of an intramedullary focus of infection with the formation of a typical abscess of the brain, associated with its invariable, localized, low-grade meningitis.

The process just outlined is affected greatly by the virulence and strain of the infecting organisms. The meningitis produced by this direct extension may prove of a marked fulminating variety, spreading rapidly throughout the whole subarachnoid space, or it may remain localized—a chronic walled-off focus of thickening. Quite similarly the local infection may not lead to necrosis and abscess formation but may remain a diffuse process without areas of true necrosis.

Spread of infection along a thrombotic blood vessel must be considered under this heading of infection by local direct extension. Likewise a possible conveyance or course of infection along a nerve sheath, particularly in the case of the optic or acoustic nerves, must be included. These varieties of direct infection present the same characteristics of pathology as do the cases spreading from foci in the air cells of the cranium.

(3) The third general method of infection within the central nervous system concerns the *introduction of septic material through perforating wounds*, or the injury, with subsequent infection, of the

brain due to extensive cranial destruction. These wounds, not of infrequent occurrence in civil life, are very numerous in the war zone. The injuries are naturally of every type from the simple scalp or cranial contusions or lacerations to the deep perforating variety. A marked difference in the seriousness of the wounds exists, dependent in some measure on whether the dura is injured. This fibrous membrane serves as a fairly efficient barrier; if not entered, the possibility of cerebral and meningeal infection is not great, with proper surgical treatment. If the dura be injured so that a passage for the infection is afforded, the possibility for local abscess formation in the brain tissue, of a localized meningitis or of a general infection is marked. Much here depends upon the virulence of the organism present, upon the promptness and care, and upon the type of surgical intervention; for the measures undertaken must be of a type not to interfere with the normal process of "walling-off" of the meningeal spaces and of the brain tissue. This becomes the more difficult when foreign bodies (bits of metal, cloth, etc.) are driven into the brain tissue, forming a definite foreign body usually of a septic nature.

When the walling-off process is examined in the light of the peculiar anatomical relations already pointed out as characteristic of the brain and its membranes, there are several complicating factors which must be considered. In the case of the penetrating wounds of the brain, the conditions are different from the spread of an infection from a focus outside of the dura (as in the middle ear). In the latter type of infection the protective walling-off process probably precedes or is synchronous with the advance of the bacterial infection. Thus in all probability there is some measure of adhesion, if not complete, between dura and arachnoid or at least within the subarachnoid space, joining pia to arachnoid. This protective mechanism (the laying down of fibrin and the inflammatory cell-wall) is probably responsible for the infection remaining a local focus and not becoming a generalized process. But in the case of the perforating intra-dural wounds, the normal protecting agencies are not called into play to prevent the spread of infection until the damage is done. Thus in the normal anatomical relationship the perforating wound tears through the dura, opens the subdural space with its capillary layer of fluid, then perforates the leptomeninges, exposing widely the subarachnoid space, releasing the cerebro-spinal fluid and affording opportunities for extensive conveyance of the infection from the zone of entry, and finally entering and destroying the brain-tissue itself.

Fortunately, the reaction of the brain to such injury is prompt and fairly efficient. The brain tissue tends to swell within a very short time, due to the development of the condition termed "cerebral edema." With this edema and with any associated hemorrhages,

the intracranial pressure increases, provided the cranial defect permits no extensive herniation of the brain-tissue.

This tendency toward herniation is the direct result of an increase in intracranial tension, and in the case of an injury, of an actual attempted increase in brain-bulk (due to edema and possible hemorrhage). The process of swelling and especially herniation is considered by several writers to be an effective means of walling-off the meningeal spaces. The mechanism by which this may be accomplished is quite simple. The herniation tends to compress the subdural and subarachnoid spaces all around the defect in the cranial vault and especially in the dura. This occasions a varying degree of injury to the cells lining the inner surface of the dura, and to the arachnoid cells. The compression thus caused around the base and edge of the herniation results in an early adhesion between the inner surface of the dura, the arachnoid and the pia. In this way the subdural space is effectually walled off from the zone of injury. Of greater importance, however, is the associated walling-off of the subarachnoid for it is in this latter complex channel that the circulation of cerebro-spinal fluid occurs, rendering easy the mechanical transport of infecting bacteria.

Other mechanisms than the mere pressure-adhesion at the base of the cerebral hernia apparently play a rôle in the walling-off of the meningeal spaces. Certainly the customary inflammatory reaction in the tissues is of utmost importance, with fibrin deposition, the wandering in of blood-cells and others with phagocytic powers, and the general reactive phenomena throughout the zone of injury. But it must be considered that this walling-off process is essential to the localization of infection; the surgical procedures undertaken must not interfere too markedly with it.

In the brain itself, the process of walling-off or of localizing an infection is not facilitated by any fascial planes or by any other dense tissues as outlined above. Here the prevention of spread of infection is solely a matter of the reaction of the tissue; this reaction may, however, be so marked in the sub-acute or chronic cases that a veritable capsule of an abscess is present. The reaction against necrosis and infection seems to follow the ordinary laws of inflammation, but in the nervous system there are some interesting possibilities of cellular activity. In addition to the ordinary blood-cells (chiefly polymorphonuclear and small or large mononuclear) combatting the infection, there is a possibility that neuroglia cells become phagocytic and assist in the process of reaction (Alzheimer). But from the surgical standpoint, the development of a definite circumscribed abscess is of importance. Subsequent measures should be undertaken with the idea of not breaking through this wall but of facilitating the growth of new granulations from it.

The cerebral wounds which penetrate deeply through the cortical zones and extend nearly into the lateral ventricles require a walling-off process, not only to protect the meningeal spaces but likewise to avoid extension of the infection directly into one of the lateral ventricles. If the initial perforation destroys or injures seriously the lining ependyma of the ventricle, infection of the ventricle may occur. The infection in such a case is largely conveyed by the cerebrospinal fluid of the ventricles; the possibility of walling-off in these cavities is not very great. The bacteria may be carried by the fluid into the subarachnoid spaces and basal cisterns, inaugurating there a posterior basilar meningitis. Associated with this basilar process, there may be a definite enlargement of the lateral ventricles, due chiefly to the pyogenic infection and formation of occluding materials. Thus in many of the perforating wounds a definite hydrocephalus with heavy pus in the ventricles may be found.

But it must be granted that if the virulence of the infecting organism be great enough within the meninges or nervous system, the walling-off phenomena, just detailed, do not suffice to prevent the spread of infection. This is true of infections in other parts of the body; if the virulence of the infecting organism be sufficient, the natural protective mechanisms of the body break down and the infection becomes systemic. Experimentally in perforating wounds of the cranium, the infection, if produced by virulent organisms, may not remain local, but may spread rapidly from the local point into an extensive meningitis of the whole hemisphere, with death in from twenty-four to forty-eight hours. In such cases, the spread is usually localized to one hemisphere, but apparently no hindrance to the spread of infection elsewhere is efficient. Surgical intervention in these cases must be prompt and must attempt to localize the infection.

Pathology of brain abscesses.—It is practically impossible to detail any characteristic pathology of abscesses of the brain, because of the varying etiological factors, all of which modify so strikingly the resultant pictures. Many of the peculiar reactions have already been commented upon in the preceding sections, with discussion regarding the general processes of localization of an infection of the brain to a limited focus.

Cerebral abscesses may be either solitary or multiple, the latter being usually small and of hematogenous origin. In the central portion of an abscess occurs a varying amount of pus, the character of which varies markedly in the acute or chronic cases, and is of no particular importance. The bacteria found in these are for the most part of the same strain as that causing the original more or less distant infection, but a secondary invader may be recovered either in pure culture or mixed with the primary organism. Such mixed infec-

tions are not uncommon. The staphylococcus and streptococcus are the bacteria commonly found. The bacillus typhosus, the bacillus coli communis, and others have been isolated in special cases.

The location of the abscess in the brain has no significance particularly, except in those cases in which it results from a direct extension of an infective process. The form of a solitary abscess varies considerably, being usually pear-shaped or possibly multilocular. The pear-shaped character of the abscess is due to the fact that the body of the abscess is usually subcortical, with constriction in the cortical zone of gray matter. The greatest destruction of tissue in consequence occurs within the white fiber tracts.

The histological composition of the abscess-wall has been studied quite extensively. In the early cases of pyogenic infection, the zone of reaction in the brain-tissue is largely made up of polymorphonuclear leucocytes. In the less acute cases mononuclear elements are found in large numbers in the reaction-wall; they are of the variety usually known as macrophages, round in shape, of large size, and usually vacuolated. These are frequently found in the subacute abscesses outside of the real "pus zone," though considerable mingling of polymorphonuclear and mononuclear cells exists. Other mononuclear elements,—the small lymphocyte, the polyblast, the plasma cell, and the "Gitterzellen,"—are quite common. In addition to numbers of these possible hematogenous cells, there is usually a large amount of fibrin in the abscess-wall, with also fibroblasts and a reacting capillary bed.

In the more chronic cases, the abscess may be completely isolated from the rest of the brain substance by a dense membrane of true connective tissue. This usually requires many weeks for its formation and occurs only in those cases where the virulence of the organism remains low for a considerable period. Bramwell considers the presence of a thick, dense lining membrane evidence that the abscess has been in existence for three or four months at least. It is of interest that the fibrous tissue composing such an abscess-capsule must be derived almost entirely by proliferation of the mesodermal fibrous tissues originally occurring in the walls of the cerebral vessels.

The more chronic type of abscess is usually the result of direct extension from middle ear disease or from an infected air sinus. On the other hand, the abscess from a perforating wound is usually of the acute type, with either a cure by drainage or an infection spreading into the lateral ventricles or into the subarachnoid spaces.

Symptoms of brain abscesses.—The symptoms in abscess of the brain are most variable. There are no absolute signs. Brain abscess is a condition to be looked for after injury to the brain or when symptoms of deep cranial injury occur, along with a chronic infection of the cranial bones or of one of the adjacent air-sinuses. The

symptoms in these cases are frequently slight, fever, followed often by a slow pulse and subnormal temperature, headache, nausea, vomiting, drowsiness, mental dulness, defective memory, irritability and leucocytosis. There may be deep-seated tenderness over the site of the abscess.

Abscesses, however, may remain latent for years; the patient may become insane, without the recognition clinically of the abscess.

Abscesses of the cerebellum may be suspected from the appearance of subcortical pain, vertigo, cerebellar asynergy, and nystagmus. Stiffness of the neck may be present in these cases. The prognosis of brain abscesses without operation is bad. In the hands of the most experienced the mortality for operations on brain abscesses is very high. Opening and free drainage are indicated. The procedure is a serious one, as fatal meningitis frequently results.

The *treatment* of brain abscess is discussed in the chapter on Gunshot Injuries, Chapter V.

CHAPTER XII.

SYPHILIS OF THE NERVOUS SYSTEM.

It is of the utmost importance to make the diagnosis of syphilitic lesions of the nervous system as early as possible, for it is in the primary stages that therapeutic measures are of most benefit. That the diagnosis of such lesions presents many difficulties is evident, since there is no part of the nervous system that may not be attacked. Certain symptoms are present, however, in nearly all cases, and it is principally by the recognition of these that we are able to make a proper diagnosis. To better understand what symptoms are possible in syphilis, an effort will be made to trace the course and nature of the disease and the method of involvement of the nervous system.

Method of infection of nervous system.—In only a few of the large number of persons affected does the nervous system become secondarily diseased. The important question, then, is: Why should the nervous system of one person and not of another be affected, and what influence, if any, does treatment have upon the prevention of such involvement?

Repeated instances have been recorded in which a number of persons, not at all related, have become infected from the same source, and subsequently developed general syphilis, and later, specific disease of the nervous system. Here are two possibilities: *First*, the most probable, that the specific infection was of such character that it had a special affinity for these structures; and, *second*, that there was lessened resistance of the nervous system to the infection. It has always been one of the old theories that only certain forms of specific intoxication will cause disease of the nervous system; the best proofs of this have been such instances as quoted above. That such is the case there can be no doubt, but such a theory will hardly answer for all cases. There is no question that specific manifestations at the present time are not as severe as they have been. This is due partially to the better recognition and earlier and better treatment, and also to the fact that all diseases have a tendency to lessen in intensity in the course of time.

It is probable that the most important factor which determines whether the infected person will have future involvement of the nervous system is the lessened resistance of these structures, or possibly a special predisposition. Just what determines this is, of course, difficult to tell, but it must be that certain parts of the anatomy are less resistant than others. Hereditary and family influences contribute to this to a large degree. Erb records instances of specific infection in families in which all had tabes dorsalis following infection from the same source.

Frequency of disease.—It is important to know in what percentage of cases of syphilis the nervous system becomes diseased. This can never be determined with accuracy, and we must rely principally upon statistics to be obtained from institutions. Erb, for instance, collected some years ago the histories of many cases of specific infection, and of these, in about 1 to 5 per cent tabes followed. In how many, however, there were other involvements of the nervous system is not recorded, but judging from the fact that tabes is no more frequent than other cerebrospinal diseases, it is probable that a fair percentage would be about from five to ten cases in one hundred. This, of course, is only conjecture.

It is also a question whether or not early treatment of the disease will prevent such future involvement. That it does so in the majority of instances there is no doubt, but in many it is of no avail. It is, however, in the more chronic cases, or in those in which the symptoms of cerebrospinal involvement appear many years after the infection, that this question arises. In many, antero-specific treatment had been instituted and carried out vigorously, and the patient pronounced well, only to have some part of the nervous system become diseased years afterwards. The conclusion is forced upon us that in some forms of syphilis the cerebrospinal system will become diseased whether or not treatment is instituted, and no matter how vigorously.

After an infection the nervous system may be involved almost immediately in the midst of treatment, or may not for some years, sometimes as many as twenty. It is generally supposed that the earlier the cerebrospinal symptoms appear, the more severe the infection. Considering, however, that the symptoms produced years after the infection are about of the same severity and character as they are in the early periods, there is probably little difference.

Pathology.—Pathologically, in the early forms, no matter where the nervous system is involved, there is always a round-cell infiltration about the bloodvessels and in the pia, this being true of the base of the brain and the posterior part of the spinal cord; and there is also a secondary endarteritis and permanent thickening of the meninges. Rarely, small gummata will be found, but these are not as frequent in the nervous system as is commonly supposed. More rarely, there is also an involvement of the substance of the cord or brain. When such is the case, in the former there is very often an extensive inflammation, this resulting in a myelitis; if in the brain, the cortex and subcortex are diseased, this causing the symptoms of diffuse encephalitis. It is characteristic of these acute inflammations, such as myelitis and encephalitis, that the symptoms appear suddenly.

When the disease comes on many years after the infection, the usual pathologic findings consist in a sclerosis of the vessels and thickening of the meninges, and sometimes in gummata, but is principally to the results of the endarteritis that the symptoms are due. There are, however, other forms of disease resulting from specific infection which are caused, not by inflammation, but by gradual changes in the nervous structures, thought to be the result of toxins. These changes mostly occur in structures which have more or less functional relation, and are called systemic, the best example of this being locomotor ataxia or tabes dorsalis.

Symptoms.—It can be readily understood, then, that, so far as the symptoms of specific disease of the nervous system are concerned, the majority of cases will show an involvement of both the brain and spinal cord, and that the focal symptoms will depend upon what particular portions of these structures are most diseased. No matter what the extent of the affection, there is in every case an infiltration of the meninges, and this in itself will nearly always give certain symptoms. There is a tendency, however, for certain forms of involvement in those cases in which the symptoms manifest themselves within a few years after the infection, and these will be discussed first.

Early Symptoms—Myelitis.—Among the earliest is a myelitis, this usually coming on suddenly, and in most instances affecting the lower thoracic or thoraco-lumbar region, and often giving the symptoms of a complete transverse myelitis, there being loss of power in both lower limbs, of the bladder and rectal functions, and of sensation. In the majority of cases some return of power can be hoped for, especially if vigorous treatment is promptly instituted. Added to the symptoms of the myelitis there will be those of meningeal involvement, such as girdle sense and pain.

Cerebrospinal involvement.—Other common early manifestations sometimes appearing in the midst of treatment, are those the result of multiple lesions of the brain and spinal cord, giving diffuse symptoms depending upon the location of the lesion. There will nearly always be motor and sensory involvement of irregular character, with the addition of meningeal symptoms of the base of the brain and of the cord.

Hemiplegia.—Hemiplegia is also common in early syphilis, this resulting from early endarteritis. In fact, hemiplegia occurring in young adults below forty years of age is nearly always syphilitic in origin; but the most common manifestations of syphilis are those the result of meningeal involvement of the base of the brain or of the spinal cord.

Meningitis.—Meningitis of the base of the brain is nearly always greatest around the chiasm, and it is because of this that the optic,

oculomotor, and sixth nerves are commonly diseased in syphilis. Whether it is because of this involvement of the optic nerves, or because the specific toxin has a special predilection for the iridic muscles, disturbance in the reactions of the pupils, with irregularities in their margins, are the most common and constant symptoms of syphilis. Such general diseases as syphilis nearly always affect those structures which are most commonly used, and it is, perhaps, because of this that the iridic muscles are so early diseased. In most instances there will be found irregularities in the margins, with a loss of the reaction of the pupils both to light and to movement of the eyeballs, and sometimes in the later stages a failure of response of the pupils to light, with preservation of movement of the eyeballs or the Argyll-Robertson pupil. There is no disease which will so constantly give these symptoms, and there are no symptoms which are so constant or which can be so much depended upon.

Oculomotor palsy, unilateral in type, occurs most frequently in syphilis; in fact, it has been termed its sign manual. Of the cranial nerves, however, the *sixth*, which is probably the most frequently diseased, causes double vision. This, as a rule, lasts only a very short time, appearing and reappearing, finally becoming permanent. Paralysis of any of the other cranial nerves is a rare manifestation. *Meningeal involvement in the spinal cord* is nearly always greatest in its posterior portion. Because of this, girdle sense and pains referred to various parts of the limbs are common.

Late symptoms of syphilis which become manifest many years after the primary infection, that is, after the fifth year, while indicating an involvement of both the brain and cord, nearly always show a preponderance of the disease in one of these structures. When the brain is mostly involved, there will be, besides the irregularity of the pupils and the slowness of reactions to light and movement, other symptoms depending upon the location of the lesion. These may consist in a gummatous deposit in any portion of the brain, or of a basal infiltration, in addition to those diseases resulting from the early endarteritis.

It is characteristic of brain symptoms, just as it is of those which are the result of basal lesions, that they may appear for a time and then disappear, only to reappear again and remain more or less permanently. In those instances in which the spinal cord is mostly involved there may be diffuse symptoms, but in nearly all there is some meningitis, causing girdle sense and pains.

Disturbances in bladder and rectal functions, especially the former, are very common in spinal infections, and sometimes may be the only symptoms indicating such disease.

Cases are observed in which there seems to be an equal involvement of the brain and the spinal cord, but even here one of these structures

may be more diseased. The symptoms will be multiple, as a rule, and there will be, besides the specific pupillary phenomena, disturbance of mentality, irregular motor and sensory symptoms, and involvement of the bladder and rectum.

So far, only those cerebrospinal syphilitic diseases have been discussed which are the result of the direct pathologic processes produced by such infection, while other diseases, as general paresis, tabes dorsalis, and postero-lateral sclerosis, are supposedly produced by its toxins. Their symptoms, as a rule, come on many years after the infection, at least so far as their appreciation is concerned. It is curious that a person may be infected with syphilis and have no symptoms for a number of years, and then may develop posterior sclerosis. This does not seem logical, and it is probable that if our methods of observation were better we should be able to demonstrate some symptoms, for the syphilitic toxin does cause early and constant changes.

Serum tests for syphilis.—Wassermann reaction. Wassermann, in 1905, described a reaction, known by his name, which is of the greatest help in the diagnosis of syphilis. Since the original descriptions many modifications have been made, the most important being by Noguchi. At the present time these tests are cumbersome and can be made only by the experienced laboratory worker. In view of this fact, and also because the technic is constantly changing, it has been deemed inadvisable to give a description of such tests, but only to discuss its significance and applicability to therapeutic measures.

These tests can be applied both to the blood and to the contents of the cerebrospinal fluid, and while it is possible to give an opinion in reactions applied to either, it is important, if possible, to give consideration to the cellular elements in the cerebrospinal fluid, the globulin content, and the specific reaction of blood and serum found in the spinal fluid.

It must be understood that there is not always parallelism between the conditions found in the blood and spinal fluid or between the Wassermann reaction and the lymphocytosis of the cerebrospinal fluid. At times the reactions are alike, but not always.

In brief, it is the present opinion that a positive Wassermann reaction means not only tabes, general paresis, or any of the chronic syphilitic diseases, but it also indicates active syphilis. This reaction has been obtained in a number of instances in leprosy, in sleeping sickness, and in other diseases, but this by no means lessens its value.

It must be remembered that the application of Salvarsan and mercury negatives the reaction, whereas potassium iodid has no such influence, this being an important indication of the value of mercurial treatment in syphilitic diseases.

CHAPTER XIII.

NASAL ACCESSORY SINUSES—THE MASTOID PROCESS.

ANATOMICAL.

The nasal cavities are in a large measure surrounded by pneumatic spaces. Laterally are the large maxillary sinuses, above are the ethmoid labyrinths, above and in front are the frontal sinuses, and above and behind are located the sphenoid sinuses. The anatomical relations of these accessory sinuses have many points of interest, such as their relation to the exterior of the skull, their relation to the interior of the skull, their relation to the orbit and to the nasal fossae.

The external relation of the frontal sinus is the most conspicuous, as this sinus is placed in a large measure between the perpendicular plates of the frontal bone. The extent of this exposure of the outer wall of the frontal sinus varies widely, depending on the size of the sinus. In extreme cases the cavity reaches to the outer angle of the orbit and upward to the roots of the hair. Small frontal sinuses are not uncommon where only a small cell is found between the perpendicular plates of the frontal bone above the inner angle of the eye. Complete absence of a frontal sinus on one or both sides is also quite common. The anterior wall of the superior maxilla forms the outer wall of the antrum or maxillary sinus, which extends from the orbit above to the alveolar process below. The ethmoid cells have an important relation to the exterior of the skull. This is just above the inner canthus of the eye. It is at this point where the sensations of pain and discomfort are located in ethmoid disease, and it is here that the superficial evidence of the rupture of an ethmoid abscess makes its appearance.

Intracranially the posterior wall of the frontal sinus is in relation to the anterior cranial fossa. It is not uncommon for the frontal sinus to extend posteriorly above the orbit. The roof of this extension is formed by the floor of the anterior fossa of the skull. The ethmoid cells lie immediately beneath the floor of the anterior fossa, just external to the cribriform plate, from which the cells are kept separated by the median plate of the labyrinth. The intracranial relations of the sphenoid sinus are of especial interest. Anteriorly the

roof of the sphenoid is formed by the same plate which forms the roof of the ethmoid, and is in this region in relation to the structures of the anterior fossa. The upper posterior wall of the sphenoid sinus is formed by the sella turcica. The sphenoidal sinus is adjacent to the cavernous sinus, the internal carotid artery, and and the optic nerves.

The relation of the accessory sinuses to the orbit is of special im-

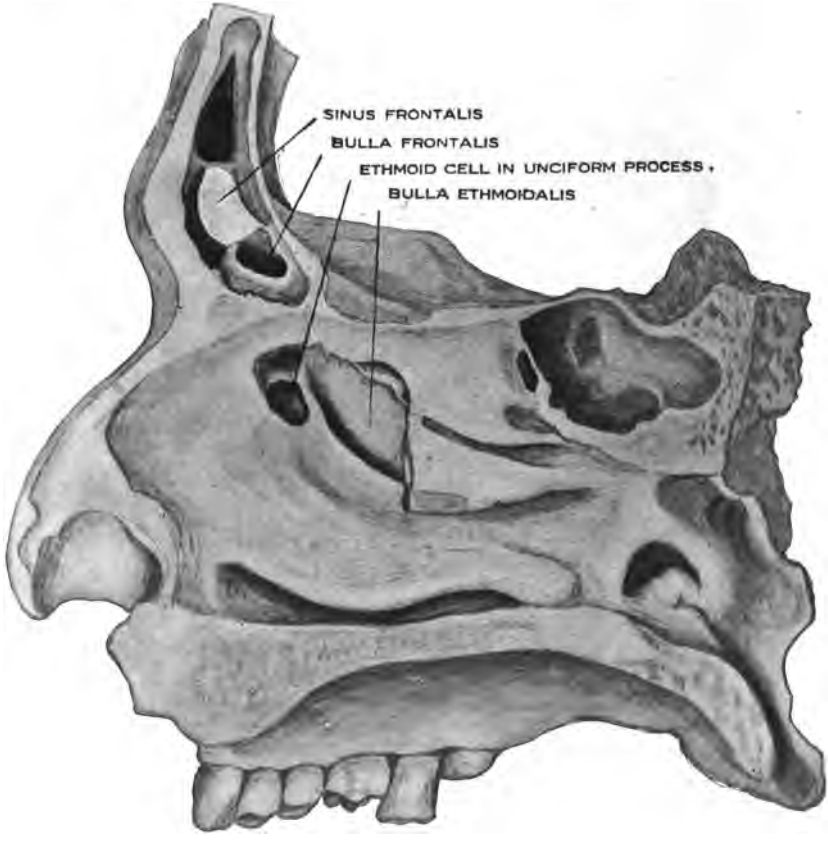


FIG. 207.—Sagittal section through the nose, showing anterior half of the middle turbinated body removed. Illustration shows the frontal sinus, the nasal aspect of the ethmoid labyrinth, and the sphenoid sinus. (Shambaugh.)

portance. The floor of the orbit forms the roof of the maxillary sinus. Dehiscence in this bony plate sometimes exists, and an empyema of the maxillary sinus with impaired drainage into the nose may result in infection of the structures in the orbit. The roof of the orbit forms the floor of the frontal sinus. In cases where there is a horizontal extension of the sinus above the eye, this relation to the orbit may be quite extensive. This wall of the frontal sinus is more frail than either the external or posterior walls. The relations

of the ethmoid sinus to the orbit are of special interest, because the most frequent involvement of the orbit from accessory sinus disease is the result of extension of disease from the ethmoid. The lateral wall of the ethmoid labyrinth is formed by the median wall of the orbit. This plate, because of its frail character, is known as the paper plate of the ethmoid and is much more delicate than the roof. A rupture of an ethmoid abscess usually takes place through this orbi-

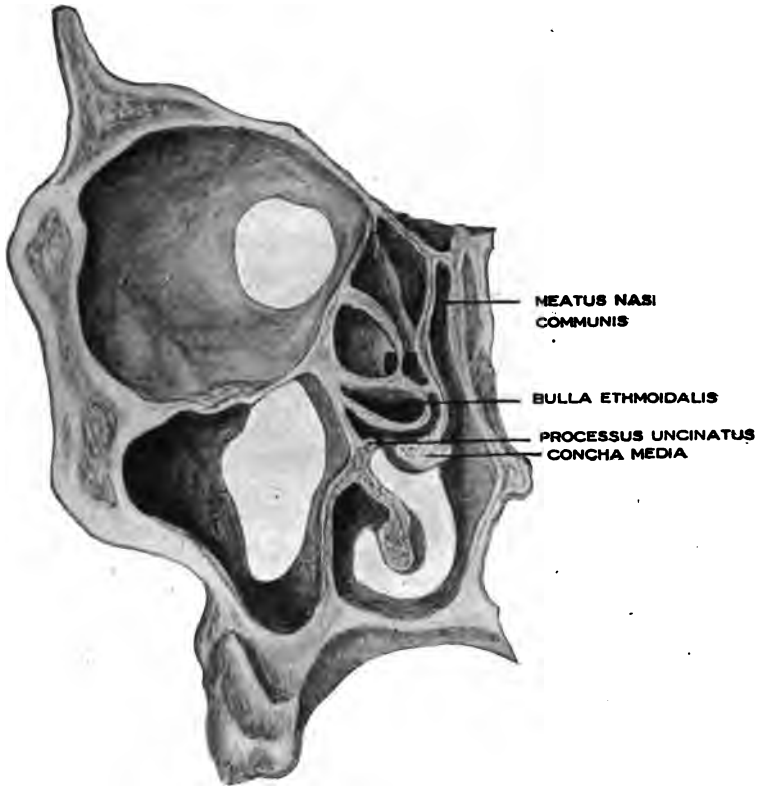


FIG. 208.—Frontal section through the head, showing cross section of the middle and inferior turbinated bodies and separation of the nasal chamber into the inferior meatus, middle meatus, and common nasal meatus. The relation of the maxillary sinus to the floor of the orbit is shown, also the relation of the ethmoid labyrinth to the orbit. (Shambaugh.)

tal wall, with the swelling presenting just above the inner canthus. The sphenoid sinus also encroaches on the orbit. The lateral wall of this sinus at its upper anterior part is formed by the median wall of the orbit. The optic nerve comes into close relation with the cavity of the sphenoid sinus in this region. This nerve also comes into close relation in this same region to the posterior ethmoid cells. An optic neuritis may occur as the result of infection either in the sphenoid sinus or in the posterior ethmoid cells.

The relation of the several accessory sinuses to the nasal cavity is of special interest, not only in making a diagnosis of sinus infection but also in connection with its treatment, which in most cases depends upon securing adequate drainage into the nose. In order to understand these relations one must keep in mind the relation of the structures forming the outer wall of the nasal chamber. In the sagittal section, one sees that the outer wall of the nose is divided by two conspicuous structures, the inferior and middle turbinated bodies, into three well-marked fossæ. The space below the inferior turbinal

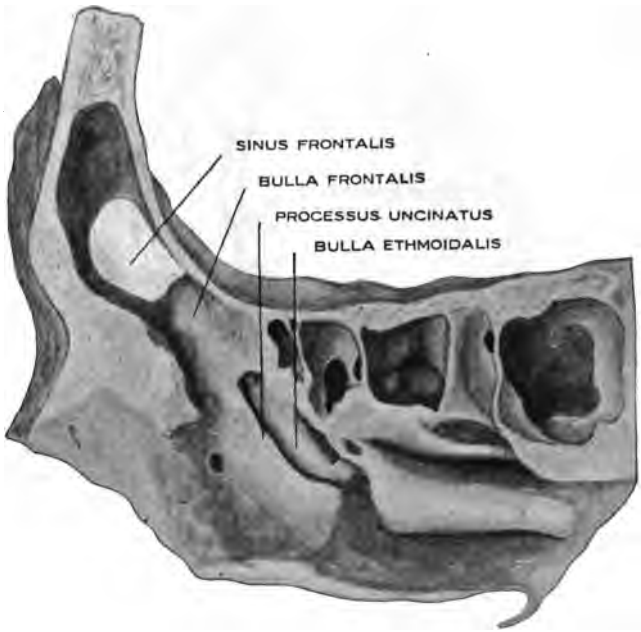


FIG. 209.—Frontal section through the head with the median wall of the ethmoid cells removed. Anterior half of the middle turbinated body has been resected. Structures found in middle meatus are shown, as well as the cavities of the frontal and sphenoid sinus. (Shambaugh.)

is known as the inferior meatus, that between the lower turbinal and the attachment of the middle turbinal as the middle meatus, and the space between the middle turbinal and the septum of the nose as the common meatus, or the olfactory space. These relations are shown very clearly in a frontal section. Three of the nasal accessory sinuses have their normal openings in the middle meatus. These are the maxillary and frontal sinuses and several ethmoid cells which may be grouped as the anterior ethmoid cells.

In the space between the middle turbinated body and the septum are the openings for two of the nasal sinuses, the sphenoidal sinus, and the posterior group of ethmoid cells.

Infection of any one of the anterior group of accessory sinuses, those which drain into the middle meatus, can usually be detected by the presence of pathological secretion, making its appearance under the free edge of the middle turbinated body. Infection of the posterior group of sinuses in like manner can, as a rule, be detected by the presence of pus appearing between the middle turbinated body and the nasal septum. A narrow nasal chamber or a high deflection of the nasal septum frequently crowds the free edge of the middle turbinated body against the outer wall of the nose in such a way as

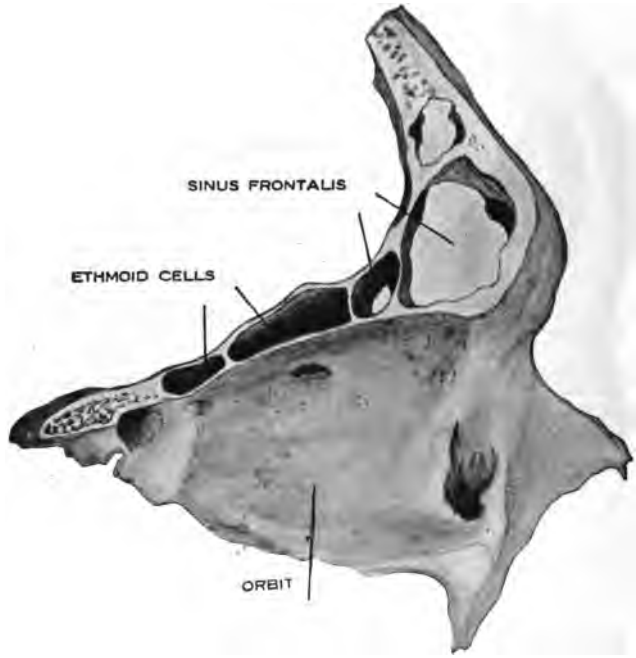


FIG. 210.—Sagittal section through the roof of the orbit, showing relation of frontal sinus and ethmoid cells. (Shambaugh.)

to interfere seriously with the drainage of the sinuses opening into the middle meatus. The same obstruction may result from an enlarged middle turbinated body. Drainage from the posterior series of sinuses is less often interfered with because of such anatomical variations.

Acute sinus infection.—Acute infection of one or more of the nasal accessory sinuses occurs in a great many cases of head colds. The symptoms from such involvement vary widely, from a sensation of fullness in the head, and discomfort, to one of very severe pain. In the milder cases and those where the drainage of the sinuses is not seriously impaired, the chief symptom is usually one of dull headache, often associated with a sensation of pressure between the eyes.

When the drainage is much interfered with, the headache may be most severe. There is nothing specific, as a rule, in the location of a headache by which the involvement of a particular sinus can be positively diagnosed. Disease of the frontal sinus produces frontal headache, but frontal pain may be the chief symptom of an involvement of other accessory sinuses, for example, the maxillary sinus or the sphenoid. Pain in the orbit is especially likely to occur from an involvement of the ethmoid cells, and less frequently from disease of the sphenoid. Occipital headache appears to be more character-

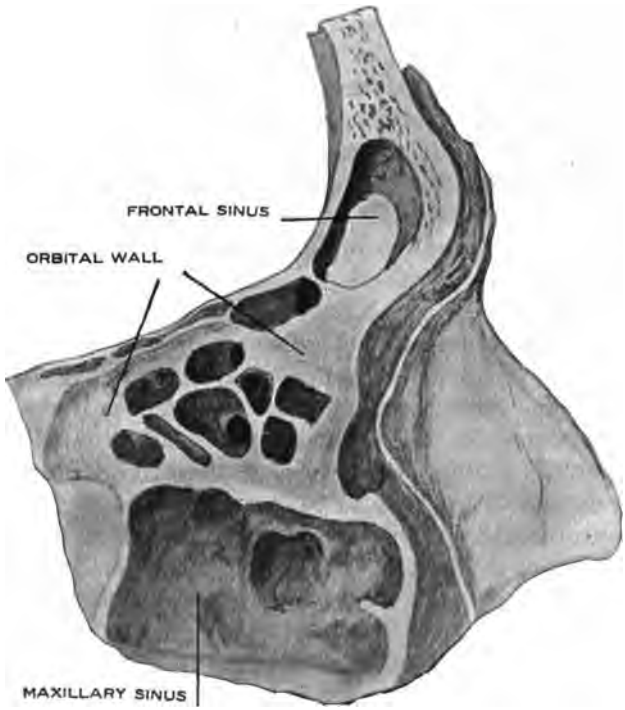


FIG. 211.—Sagittal section through the orbit. The orbital wall of the ethmoid cells has been removed. Intimate relation of ethmoid cells to the orbit is shown. (Shambaugh.)

istic of sphenoid sinus trouble than for any other type of sinus disease. Sensitive upper molar teeth are quite characteristic of an acute maxillary sinus infection, even when this condition is the result of extension from the nose and not primarily from a molar root. Tenderness on pressure over the anterior wall of the maxillary sinus is rarely absent when this cavity is the seat of an acute infection. The same is true of the region over the frontal sinus in acute frontal sinus disease. When the anterior ethmoid cells are involved, the region at the upper inner margin of the orbit is usually quite sensitive to pressure.

Intranasally, the symptoms of acute sinus infection are, as a rule, quite characteristic. This consists of a swelling of the soft tissues and a greater impairment of nasal respiration on the involved side of the nose. A unilateral increase of discharge from the nose is also quite characteristic of a one-sided infection, provided the drainage of the affected sinus is not seriously impaired. The diagnosis of the particular sinus involved can usually be assisted by a careful intranasal examination. This examination is made by first clearing the nasal chambers of all secretion, then applying a weak solution of cocain (1 per cent) to the swollen mucous membrane, especially about the middle turbinated body. If pus reappears in the middle meatus, it is evident that the trouble lies in one or more of the anterior group of nasal sinuses, the maxillary sinus, the frontal sinus, and the anterior ethmoid cells. Transillumination of the maxillary and frontal sinuses will, as a rule, suffice, together with the external condition over the sinus, and the intranasal findings to determine the diagnosis. The skiagraph is of especial assistance, particularly in the case of the frontal sinus, as a shadow over this sinus by transillumination may be the result of an anatomical variation in the size of the sinus, as well as of pathological conditions within the sinus. The skiagraph gives not only an accurate outline of the shape and size of this cavity, but will also, as a rule, show very clearly whether the frontal sinus is diseased. The transillumination test as well as the skiagraph is of much less assistance in making a diagnosis of ethmoid disease, but since the anterior ethmoid cells are rarely involved, except in association with disease of the frontal or maxillary sinuses, and especially since the intranasal treatment of the latter relieves as well the pathological conditions in the ethmoid cells, it is not a matter of any great moment not to be able always to diagnose more accurately the existence of ethmoid disease.

When an acute inflammation is limited to the posterior series of accessory sinuses, that is, to the sphenoid sinus and the posterior ethmoid cells, the intranasal findings are also quite characteristic but, on the whole, somewhat more difficult of recognition. It will be necessary, as a rule, to cocainize thoroughly the middle turbinated body so as to secure as much shrinking of the soft tissue as possible. Pathological secretion reappearing between the septum and middle turbinated body can come only from disease of the sphenoid or posterior ethmoid cells. This diagnosis can often be facilitated by tipping the head forward in order to empty secretion from the sphenoid sinus, the opening of which lies near the top. The skiagraph, though not so reliable, is nevertheless of distinct value in making a diagnosis of sphenoid sinus disease in cases where the result of intra-nasal examination still leaves one in doubt.

The treatment of acute sinus infection is, as a rule, quite simple, and consists for the most part of measures aimed to secure better drainage from the normal openings. In most of the cases this can be accomplished by intranasal applications to the swollen membranes. Cleansing the nasal chambers by means of a warm alkaline nasal douche, followed by the application of a 1 per cent cocain solution made up in a solution of adrenalin, 1 to 10,000, applied by means of a cotton swab to the region of the middle turbinated body is, as a rule, all that is necessary. This may be repeated a couple of times during the day. In the more severe cases of maxillary sinus infection, especially where the severe symptoms persist over several days, irrigation of the sinus by puncturing through the inferior or middle meatus will be of great assistance. In these severe cases, especially where the frontal sinus is involved and where sufficient relief can not be obtained by the simple intranasal treatment outlined above, surgical measures should be taken to secure better intranasal drainage. This is accomplished by the removal of the anterior end of the middle turbinated body, followed, if necessary, by exenteration of the anterior ethmoid cells, and the enlargement of the normal opening into the frontal sinus. In cases where the maxillary sinus is also involved, the operation may include an opening into this sinus by breaking through its nasal wall in the region of the middle meatus. When the posterior series of accessory cells is involved and local application fails to bring relief, exenteration of the posterior ethmoid cells, followed by a removal of a part of the anterior wall of the sphenoid sinus, may be required. An external operation for the relief of acute sinus disease is never called for in the case of the maxillary sinus, and very rarely in the case of the frontal sinus. Only in cases where intranasal methods fail to secure an opening into the frontal sinus and where the persistence of severe pain makes further operative measures imperative is one justified in resorting to an external operation.

Chronic disease of the nasal sinuses.—The symptoms of the chronic diseases of the nasal sinuses are quite similar to those of the acute. The most conspicuous symptom is a profuse purulent nasal discharge, which, when unilateral, is particularly suggestive of an accessory sinus disease. The diagnosis of the particular sinus involved is made, just as in the acute cases, by a careful intranasal examination supplemented by the transillumination tests of the frontal and maxillary sinuses and by the use of the skiagraph in doubtful cases.

The treatment is always surgical. The aim should be to secure adequate drainage and ventilation of the involved sinuses. Radical exenteration of the affected sinuses, especially of the maxillary and frontal sinuses, has now been given up as a routine measure in these chronic cases. Radical exenteration of the ethmoid cells is still

practiced, as it seems to be the only feasible method of eliminating chronic suppuration of these cells. When the disease is limited to the maxilllary sinus, all that is required to accomplish a cure is to secure a large opening into the nose. This can be made most readily in the middle meatus. It is usually desirable to remove the anterior part of the middle turbinated body as a preliminary step. With this obstruction out of the way, it is the work of but a few moments to make an opening into the antrum in the middle meatus one-half inch broad by three-quarters of an inch long. No subsequent treatment is required, as the ventilation of the sinus, secured by this means, will cure all cases where infection is not continued because of disease of the bone, such as accompanies an infection about the roots of upper molars. Many operators, however, prefer to drain the antrum below the inferior turbinate.

The drainage and ventilation of the frontal sinus in chronic cases is also accomplished by intranasal measures. The operation consists of the removal of the anterior part of the middle turbinated body and exenteration of the anterior ethmoid cells, followed by an enlargement of the naso-frontal duct by means of frontal sinus rasps. This treatment will suffice to cure many chronic cases, and, of the cases which are not cured by these measures, most of them are relieved of discomfort and pain as well as of the danger of such complications as may follow the retention of pus in the sinus. An external operation on the frontal sinus in chronic cases is necessary only in the cases where complete painstaking intranasal work has failed to secure adequate drainage and where the persistence of severe pain makes such an operation imperative.

In cases of chronic infection of the sphenoid sinus the problem of treatment is, on the whole, much simpler than in the case of the frontal sinus. The operative result, however, is often hard to maintain.

The indication here is met by the removal of the anterior wall of the sphenoid sinus by enlarging the normal opening. In many cases this operation will have to follow an exenteration of the posterior ethmoid cells.

Owing to the intimate anatomical relationship between the accessory sinuses and the cranial cavity there is always a possibility of the spread of infection from the sinuses to the coverings of the brain or to the substance of the brain itself. Every large surgical clinic occasionally has such cases. They are rare, however, in the practice of any one individual. Meningitis occurs most often after disease of the sphenoid and manifests itself by a rapidly fatal basilar meningitis. The frontal sinus ranks next as a cause of intracranial complications. It is the only one of the accessory sinuses which seems to

give chronic brain abscess. The ethmoid cells from their position and structure should transmit infection more often than the other accessory sinuses. Proved cases of meningitis from this source, however, are rare. Operative meningitis following instrumental work in the ethmoid cells unfortunately occasionally occurs. Every surgeon faces the possibility of it when he operates in the ethmoid region. The same is true, but to a less extent, of operations on the sphenoid and on the frontal.

Acute mastoid disease.—Mastoiditis, occurring in acute otitis media, is usually the direct cause of those more serious intracranial conditions which complicate middle ear infection. Thrombosis of

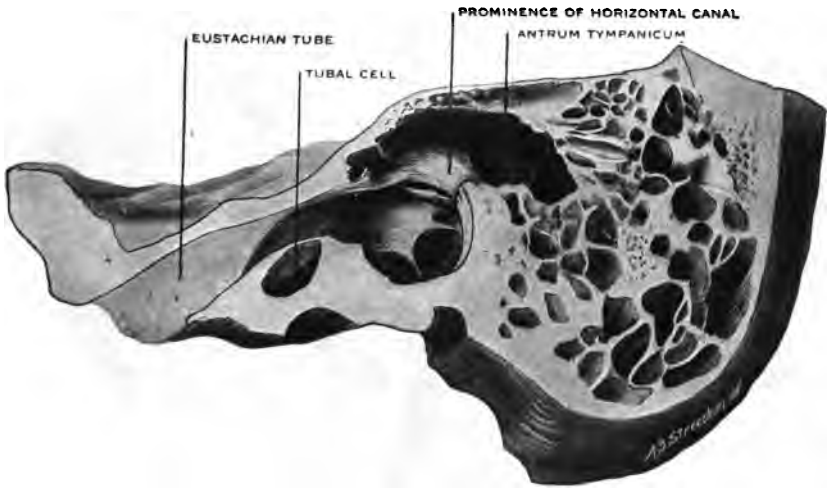


FIG. 212.—Section through temporal bone, showing Eustachian tube, tympanic cavity, the antrum, and mastoid process. Note the large cell found in front of the tympanum opening into the Eustachian tube; also note the relation of antrum to tympanic cavity. This is really one large chamber instead of two more or less separate cavities. The horizontal canal lies in the floor of the passageway from the tympanum to the antrum. (Shambaugh.)

the lateral sinus or of the bulb of the jugular, as well as meningitis or brain abscess, may, however, develop in the course of an acute otitis media quite independent of the condition which we recognize clinically as a mastoiditis. In these cases the infection extends through the roof of the tympanic cavity, causing a meningitis or a brain abscess in the middle cranial fossa, a temporo-sphenoidal abscess; or the infection extends through the floor of the tympanum to the bulb of the jugular, producing sinus thrombosis. A suppuration of the labyrinth may also cause either a meningitis or a cerebellar abscess. Such direct extension of infection from the cavum tympani is, however, rare. The severe complications of acute otitis media are as a rule the result of extension of the infection from a diseased mastoid process.

Mastoiditis, as a complication of acute otitis media, is extremely common. Acute mastoiditis which requires surgical interference is, however, relatively uncommon. In the treatment of these cases it is of great importance that one keep clearly in mind a few fundamental facts in the pathology of acute mastoiditis. The first is that acute suppurative otitis media does not occur as a disease of the tympanic cavity independent of the pneumatic spaces in the mastoid process. The antrum is anatomically but a part of the tympanum and is always involved in the acute inflammatory processes which invade the tympanum. The mastoid cells rarely, if ever, escape entirely being involved in the acute processes which infect the

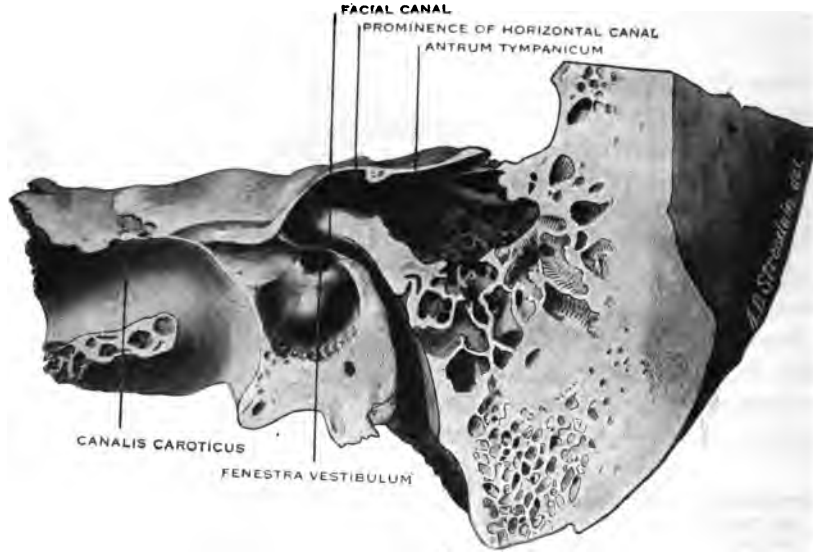


FIG. 213.—Cross section through the temporal bone, showing the tympanic cavity, the antrum, and mastoid process. The canal for the facial nerve has been exposed from the point where it enters the tympanum to the stylomastoid opening. The frail character of the shelf of bone separating the tympanum and antrum from the middle brain fossa is shown. The mastoid process is only partially pneumatic. The whole of the tip of the mastoid is diploetic, and does not contain mastoid cells. Note the thickness of the bone between the pneumatic spaces and the outer wall of the mastoid. (Shambaugh.)

tympanum. The presence or absence of clinical symptoms of inflammation in the mastoid process, especially of tenderness over the mastoid, is dependent largely on anatomical conditions. A pneumatic process with the usual arrangement of large cells around the margin, and especially in the tip of the mastoid, is usually more or less tender to pressure, even in a milder form of inflammation in the cells, whereas the partially pneumatic process with its usually thick cortex will present no symptoms of tenderness, even in those cases where a serious inflammation is going on in the mastoid, with softening of the bone and with abscess formation. It is evident at once,

therefore, that the recognition of a mastoiditis, especially by the presence of tenderness over the process, or even because of changes in the overlying soft structures or by means of a shadow on transillumination or in the skiagraph, is by no means an indication that a condition exists requiring surgical assistance. On the other hand, it is equally true that the absence of changes over the surface of the mastoid or of tenderness on pressure does not exclude the possibility of the presence of a serious form of mastoid disease urgently requiring surgical measures for relief.

With these fundamental facts in mind, let us inquire more closely into the symptoms of acute mastoid diseases, and especially into those symptoms which indicate a type of inflammatory process which requires surgical interference.

Symptoms over the mastoid such as pain and tenderness and even of alteration in the soft structures over the process, when they occur in the early stages of acute otitis media, that is, before the opening of the membrana tympani has been established, are not in themselves an indication for surgical interference. Mastoid symptoms at this stage of otitis media have a much less sinister significance than when these symptoms develop some days after the discharge from the middle ear has been established. The first aim in the treatment of such cases is to secure an opening of the membrana tympani as early as possible. The continuation of a severe otitis media without drainage through the tympanum is one of the frequent causes for the development of a mastoid abscess. If the drainage of the tympanum has been accomplished through the spontaneous rupture of the drum membrane it rarely requires a further opening, even where a bulging of the drum membrane exists, except in cases where the perforation closes completely. When a spontaneous rupture has not taken place promptly, an incision of the drum membrane should not be delayed. The subsequent treatment is enforced rest in bed, with hot applications over the ear and mastoid process, preferably by means of hot moist bandages, supplemented by the use of a hot-water bag or an electric pad. Gauze drains in the external canal are of no particular advantage, and may often serve as a means of retarding instead of promoting the desired drainage. Hot irrigations can usually be dispensed with, but are never a disadvantage in such cases. They are of assistance especially when the discharge is profuse and thick, and as a means of quieting the severe pain in the more acute stages.

In cases where the severe symptoms of mastoid reaction persist in spite of drainage and proper treatment without distinct evidence of abatement for six or eight days, the surgical opening of the mastoid with exenteration of the cells is indicated, even though the

temperature may show but a slight elevation. Fulminating cases do occur where the persistence of severe spontaneous pain with or without a marked elevation of the temperature makes an early operation necessary, but such cases are, after all, extremely uncommon.

The development of distinct mastoid symptoms some days after the discharge through the membrana tympani has been established, should always be regarded with suspicion, as they usually indicate

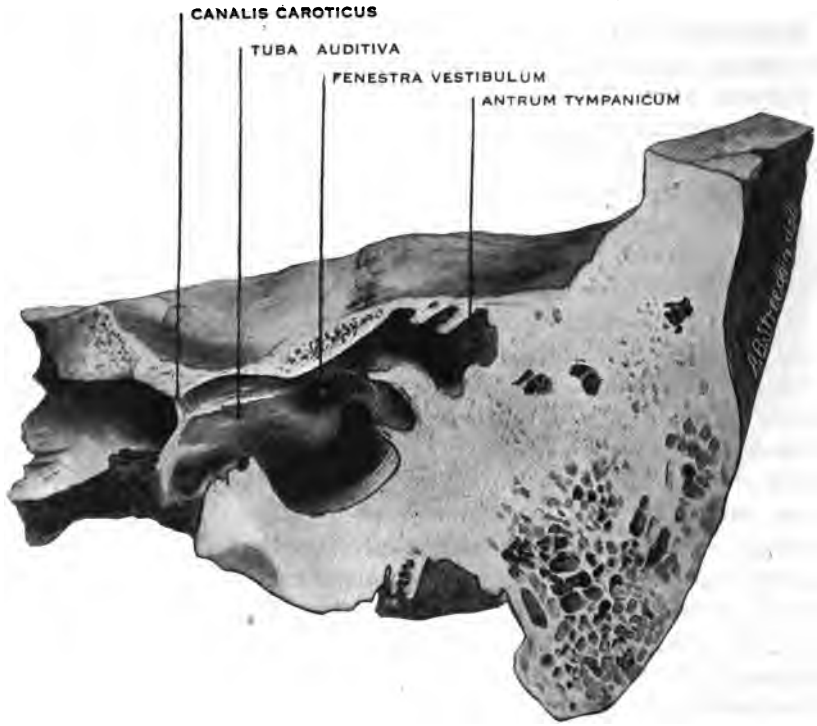


FIG. 214.—Section through the temporal bone showing tympanum and antrum. Mastoid process does not contain pneumatic spaces. This is the type of mastoid process usually found in cases of chronic suppurative otitis media, where the suppuration began in early childhood. (Shambaugh.)

the development of a softening in the bony structure of the mastoid with abscess formation. In such cases the occurrence of changes in the soft structures over the process, swelling and edema, can usually be accepted as an indication for a mastoid operation. In this connection it is important to call attention to the fact that the pneumatic spaces of the mastoid occupy the upper posterior segment of the bony external meatus. A sagging of the membrane in this part of the external meatus has exactly the same significance as does the occurrence of swelling over the mastoid.

The persistence of pain in the mastoid even in the absence of changes over the process or of tenderness on pressure should be regarded with suspicion, especially when associated with a profuse purulent discharge. A skiagraph of the mastoid in such cases may be of assistance in showing the kind of process involving the mastoid. The presence of a shadow from transillumination or in the skiagraph is not in itself significant, as this occurs in all cases where the cells are filled with pus. The skiagraph, however, will often show the presence of an abscess cavity formed by the breaking down

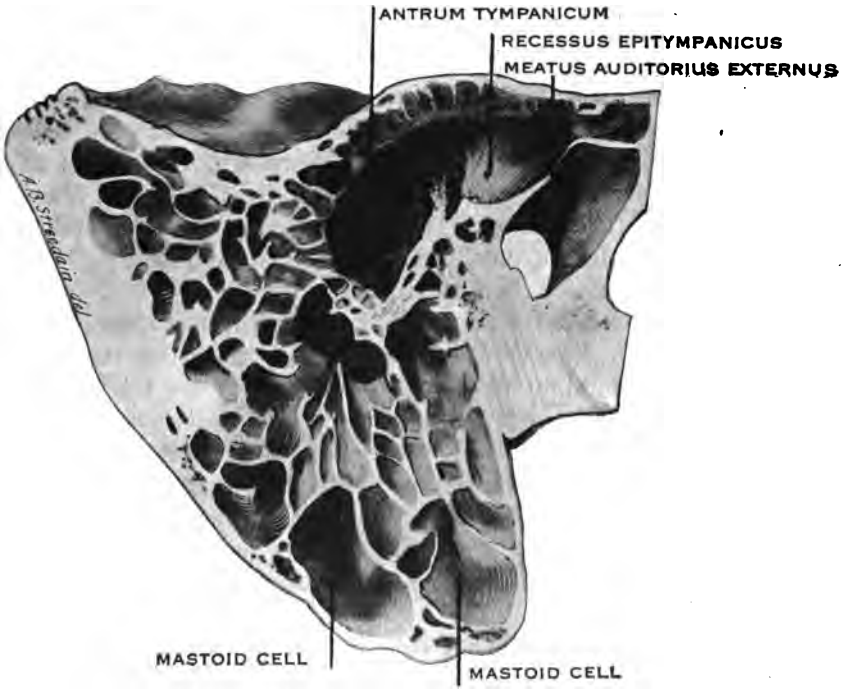


FIG. 215.—Section through the mastoid process showing the relation of the antrum to the upper posterior wall of the external canal. The mastoid is entirely pneumatic and contains no diploetic bone. Note the large pneumatic spaces at the tip of the mastoid. (Shambaugh.)

of the bony substance of the mastoid. This condition when detected always calls for a mastoid operation.

There is still another type of symptom indicating a mastoid disease with softening of the bone which should not be overlooked. This is the persistence of a profuse purulent discharge, even in the absence of pain or of changes over the process; when such a discharge continues without abatement in spite of proper treatment for three or more weeks the presence of an abscess in the mastoid bone should be suspected. Here again the skiagraph may be of assistance. It is not a mistake in such cases, even where the skiagraph leaves one in doubt

as regards the existence of an abscess formation, to check the process by an opening of the mastoid. This is important, especially since the longer continuation of the discharge of pus through the tympanum endangers the function of hearing by producing permanent inflammatory changes which interfere with the sound conducting mechanism in the tympanum. In these latter cases the rise in temperature may be quite insignificant, not more than 99° or 100° F.

It goes without saying that in these cases of acute otitis media the occurrence of more severe symptoms, such as would indicate a meningeal irritation, calls for an immediate mastoid operation. The persistence of a high temperature, 102° or higher, in an adult, if continued for several days after drainage of the tympanum, should be regarded with suspicion, and especially if other symptoms of mastoiditis are present, the process should be drained. The occurrence of a sudden rise in temperature to 104° or more, with or without the occurrence of a chill, should be taken as an indication for an immediate drainage of the mastoid. In such cases care should be taken, of course, to exclude the possibility of a chest complication being responsible for the rise in temperature.

It is not uncommon for a mastoid abscess to rupture through the digastric fossa. In these cases no change may occur over the outer surface of the mastoid, but a swelling will develop below the mastoid in the neck.

The examination of the blood in cases of acute otitis media with suspected mastoid abscess formation gives usually little assistance. The bacterial examination is often of more significance. A streptococcus infection is usually present in the more severe cases of mastoiditis which leads to intracranial complications, such as sinus thrombosis and meningitis.

In operating upon the mastoid the aim should be to open all the infected cells. The cortex of the mastoid must always be removed with caution, because of the variation in the location of the lateral sinus, which sometimes lies very close to the surface and is separated but a few millimeters from the external meatus. It is important to remove thoroughly all softened bone, but it is not desirable to curette too vigorously about the antrum except as this may be required in cases where the bone in this region has undergone softening. In the floor of the antrum lies the prominence of the horizontal canal, and immediately in front of this lies the facial nerve. Both of these structures may be injured by a single stroke of the mallet if one does not recognize the antrum cavity and continues to chisel still deeper. Curetting out the cavity of the antrum should be done only with extreme caution, and it is not advisable to attempt to enlarge the passage leading to the tympanum. Such a procedure endangers

the incus which lies in this passage. A dislocation of this ossicle will interfere seriously with the return of hearing after the subsidence of the inflammation.

Chronic mastoid disease.—Conditions occurring in chronic suppurative otitis media which will call for an operation on the mastoid are on the whole more difficult to diagnose than are the conditions requiring the mastoid operation which complicate an acute otitis media. The development of an acute mastoiditis as a complication of an acute exacerbation of a chronic suppurative otitis media does occasionally occur, and is recognized in much the same way as is mastoid disease complicating an acute otitis media. The serious complications of a chronic suppurative otitis media are the same as those which occur in acute middle ear disease. The danger of serious intracranial lesions, complicating a chronic purulent otitis media, is now well recognized. The danger of these complications is not the same in all chronic cases. Our aim here is to discuss particularly those symptoms which help to distinguish the cases which are distinctly dangerous and require an operation from those in which the disease is not particularly dangerous and which, therefore, do not require surgical treatment.

The superficial evidence of the middle ear disease, that is, the amount of the discharge from the ear, is usually of no especial significance in differentiating these cases. A profuse discharge is not infrequently present in the most harmless type of chronic otitis media. On the other hand, a discharge so slight as to be entirely unrecognized by the patient is often present in a very dangerous disease of the middle ear. The character of the discharge is, however, of great significance. A mucus-like discharge, even when quite profuse, is not found in the dangerous type of chronic otitis media except as it may occur in a temporary acute exacerbation of a chronic process. A distinctly purulent discharge, especially when associated with an offensive odor, is rarely found except in the more dangerous type. Douching of the ear which shows a distinct granular character to the washing indicates the presence of a bone-invading disease and points therefore to a dangerous type of chronic otitis media. The presence of epithelial flakes in the washings indicates the existence of a cholesteatoma, a type of process which frequently leads to serious complications.

The conditions found on examination of the external canal are of great assistance in making the diagnosis. The location of the perforation of the membrana tympani is of especial significance. A central perforation even when it involves the larger part of the drum membrane is not in itself an unfavorable sign. A perforation which

erodes the bony margin to which the membrana tympani is attached is of great significance, since this type of perforation is without exception found in the dangerous cases of otitis media. Such a perforation, however, is found also in cases where the process is not an especially serious one, and can not be taken, therefore, by itself as an indication for an operation on the mastoid. A perforation in Shrapnel's membrane is found only in cases of chronic otitis media and is not infrequently accompanied by a cholesteatoma in the attic. This is a type of cholesteatoma which can frequently be handled safely by local measures, that is, by irrigation through a suitable attic canula.

Granulations about a marginal perforation, whether this be located in the upper posterior quadrant of the membrana tympani or in Shrapnel's membrane, are unfavorable signs, especially when they are abundant and associated either with a foul-smelling discharge or evidences of a cholesteatoma.

Pain either in or about the ear should be considered carefully. The persistence of a dull pain on the affected side is often found in cases where the suppurative disease is invading the bone, that is, where a dangerous process is going on in the ear. On the other hand, pain in the ear as the result of an acute exacerbation of a chronic process has no special significance any more than has a temporary increase in the discharge in such cases.

Pathologically the diagnosis between the non-dangerous and dangerous types of chronic running ear is the diagnosis between a disease which, on the one hand, involves only the membrane lining the middle ear cavities and of a process, on the other hand, which is invading the bone, either as the result of caries or of a cholesteatoma. Clinically this differentiation can be made in almost every case, and when there is no positive evidence of a bone-invading process an operation on the mastoid is not called for. This, of course, does not mean that a relatively harmless type of chronic otitis media may not later take on a more serious character, but when it does undergo such a change—that is, when the disease extends to the underlying bone as the result of caries or because of the formation of a cholesteatoma—the evidence of such a change will appear long before an actual complication can develop. It is important, therefore, that such cases be seen occasionally by some one who knows how to differentiate between conditions which may lead to trouble and those in which a complication is not threatened. The fact that an acute exacerbation of a relatively harmless chronic otitis media may occasionally result in a serious complication is not a sufficient reason for subjecting such cases to a radical mastoid operation as a prophylactic measure.

The operation that is called for in cases where there exists a chronic discharge from the ear is quite different from the mastoid operation done for the cure of a mastoid disease complicating an acute otitis media. The operation consists of an exenteration of the diseased process not only in the mastoid but in the attic and tympanum as well. All these cavities are thrown into one large chamber by the removal of the posterior wall of the external meatus. The membranous canal is split so as to preserve this chamber, the opening back of the ear is closed primarily, and the effort is made to secure complete epidermization of the walls of the bony cavity. The operation is more difficult than that undertaken for acute mastoiditis, and there is danger of injuring several important anatomical structures when undertaken by one who has not had special training in this field of surgery.

CHAPTER XIV.

TECHNIC OF POSTMORTEM EXAMINATION, WITH SPECIAL REFERENCE TO THE NERVOUS SYSTEM.

In performing an autopsy of a neurological case it should be borne in mind that the condition of the entire body must be carefully studied before the neuro-pathological findings can be interpreted, and in selecting specimens for permanent preservation other affected organs may be as important as the brain. Especially is this true in certain cases where the sense organs, peripheral nerves and ganglia, and ductless glands present the essential pathology. Directions for the routine examination of the non-nervous organs are not given here, as they are readily available.

The methods in use for examination and preservation of the brain and spinal cord are so varied that a standard procedure which experience has shown to be adapted to the conditions prevailing in Army laboratories is here outlined. (But no single method is applicable to all cases and the judgment and experience of the pathologist will often be taxed in deciding how best to deal with the varying conditions that arise.)

The Army Medical Museum is charged with the responsibility of collecting every form of original material which illustrates the effects of injury and disease and the results of treatment. This involves the collection of material for both gross and microscopic specimens. Material sent to the Museum should be prepared with a view to permanent exhibition or intensive microscopic study. In many cases a specimen can be used in both of these ways if properly prepared. Fortunately, prompt and thorough fixation in 10 per cent formalin (4 per cent formaldehyde) is adequate for most neurological purposes, so that the procedure of preparation is simple. Pathological material loses its significance when unaccompanied by clinical data. Accordingly, every specimen should be accompanied by copies of all autopsy records and clinical histories.

In the case of the brain especially, adequate fixation can be secured only by injecting the fixing fluid through the blood vessels or by cutting the entire brain into thin slices. Slicing the fresh brain, however, results in great distortion from unequal shrinkage and interruption of continuity of fiber tracts and other structures. The cases in which formalin injection is indicated are those which may show cerebral tumors, degenerations from trauma, hemorrhage, embolism, or specific toxins, and in general all cases where extensive study of

fiber tracts may be required. On the other hand, injection of the brain before its exposure kills micro-organisms, destroys the natural color and also evidences of edema, congestion and hyperemia, and alters the normal consistency under palpation.

The excellent fixing and penetrating powers of 10 per cent formalin enable the operator to secure good results by exposure of the brain, study in situ, removal, further examination, and then immersion of the entire specimen in formalin. This is recommended as a routine procedure. After thorough fixation in formalin a wide variety of microscopic preparations can be made, including serial sections if called for.

If bacteriological and serological examinations of the brain fluids and the other steps requiring inspection of the freshly exposed surface mentioned above can be dispensed with, the simplest procedure is to open the thorax, cut the carotid arteries as low down as possible, insert the nozzle of a bulb syringe into each carotid, and inject 10 per cent formalin until it returns free from blood in the jugular veins. The thoracic and abdominal viscera may then be autopsied, after which the brain will be sufficiently hardened to permit its removal and immersion in formalin.

Another method permits bacteriological and other studies of the freshly exposed brain surface, this to be followed by formalin injection. As the first step in the autopsy, remove the calvarium and raise a flap of dura so that cultures can be made and the fresh meninges studied. The larger meningeal vessels and venous sinuses must not be opened. Then replace the scalp and inject the carotids with formalin as before. Some leakage occurs, but not enough to make proper embalming of the face impossible.

For the reasons already mentioned it is desirable that the examination of the brain (or its injection with formalin) should precede the other steps in the autopsy.

The brain.—The outline which follows is adapted for those cases in which the brain is not to be injected with formalin. The operator will, of course, make the required modifications if the brain is to be injected and for other special cases as they arise.

The cranium.—After placing a wooden block under the head, make an incision from a point behind one ear upward over the top of the head and down to a similar point behind the other ear. If, after beginning, the point of the knife is pushed under the scalp with the edge turned outward, the hair will not be cut by the incision and will serve to cover the suture. The scalp is then loosened and reflected forward and backward, dissecting it free from the cranium to avoid unnecessary stretching.

The exposed surface of the skull should be examined for fractures, atrophy, hypertrophy, softening of bone, delayed synostosis,

supernumerary bones, tumors, syphilitic or tuberculous lesions, asymmetry, previous trephining, osteomyelitis.

Now remove the calvarium with a saw by a circular cut in a plane passing around the head one-half to one inch above the glabella anteriorly, external auditory meatus laterally and just above the inion posteriorly. Care should be taken in removing the calvarium that the meninges are not sawn or torn. The calvarium is pried off with a chisel, which must be used with great care in breaking up remaining bridges of bone. In all subsequent manipulations of the brain the gentlest measures should be used, avoiding unnecessary pressure or traction. All nervous tissue that is to be examined microscopically should be handled with extreme care; otherwise post-mortem artefacts will confuse the pathological picture.

The dura.—Morbid adhesions between the dura and the skull should receive especial attention in cases of chronic alcoholism, old injuries, and sunstroke. Separation of a tightly adherent dura from the calvarium may be accomplished best with a flexible flat instrument, preferably a spatula. The dura should be examined for congestion and hyperemia, false membranes, rusty staining, granulations, degree of distention, adhesions to pia-arachnoid, fibrous felting, osteophytic metaplasia (especially in the falx). The thickness, color, and texture of the dura should be noted. Open the longitudinal sinus for its entire length with scissors and inspect its contents and lining. Cut the dura with scissors along the line of the sawed edge of the skull. Give descriptions of any extra- or intradural hemorrhages, traumatic conditions, secondary inflammations (extensions), pachymeningitis hemorrhagica.

Cerebrospinal fluid.—Examinations of the cerebrospinal fluid postmortem are never as satisfactory as those made during life. Specimens of fluid are best obtained after death by either lumbar or occipito-atlantoid puncture. If this withdrawal of fluid be done shortly after death, the cell-count and the determination of the type of cellular elements may be of value. The same methods should be employed here as given in a previous chapter as applicable to the fluid obtained during life. The greater the delay in obtaining the spinal fluid the more misleading are the data procured. This statement holds not only for the cell-counts, differentials, etc., but also for Wassermann tests, goldsols, and other more specialized reactions.

In some cases no cerebrospinal fluid can be withdrawn either by lumbar or occipito-atlantoid puncture. Such a failure to obtain fluid may be accounted for by a postmortem readjustment of the fluid relationships, at the expense of the preexisting spinal fluid.

Notation of the amount and character of cerebrospinal fluid over the hemispheres and in the base of the cranium should be made, as

well as observations of any abnormal distribution. In serous meningitis a local or generalized increase in quantity of the fluid, without other gross abnormality, will be found.

With the opening of the dura and the consequent exposure of the brain covered by the leptomeninges, certain observations should be made as soon as possible. The most important abnormalities here are those relating to infection of the meninges and brain—both local and generalized. Any possible change in the contour of the cerebral convolutions, especially flattening with obliteration of sulci, is of great importance. Such flattening of gyri may be accounted for by either an actual increase in brain-bulk (as in edema) or by an associated internal hydrocephlus.

Pia-arachnoid.—The delicate transparent pia-arachnoid quickly becomes clouded and opaque when exudate exists in the subarachnoid space. Such an inflammatory reaction causes, in the acute purulent cases, a marked creamy opacity of the membranes, with more or less distension of the channels, obliteration of the characteristic architecture, and loss of luster. In the more chronic cases this opacity of the membranes may be merely of a silvery whiteness and a mild matting together of finer structures. Definite milkiness of the pia-arachnoid, thickening or fibrous adhesions with obliteration of channels are further evidences of a chronic infection. Cultures and smears from the subarachnoid space are of value in both acute and chronic processes.

In addition to the indications of meningeal infection the pia-arachnoid should be examined for hemorrhages; these should be differentiated from those of the brain substance. Small surface petechiae throughout the membrane may occur, as also more extensive extravasations, especially following cranial injuries. A hemorrhagic variety of meningitis frequently occurs in the acute fulminating infections. Dilatation of blood vessels, either local or diffuse, may give aid in the diagnosis of infection. Old hemorrhage frequently results in a slight staining (rusting) of the membrane.

The vessels in the pia-arachnoid may be examined for evidences of emboli (with associated areas of cerebral softening), of thrombosis and of atheromatous changes.

Removal of brain.—Detach the frontal dura from the cranium, cut the tentorium cerebelli on each side, and gently raise the frontal lobes of the brain with the left hand. Then cut the olfactory, optic, and other nerves as close to their foramina as possible. The infundibulum may be divided with a knife and the hypophysis removed subsequently. After cutting all nerve roots, lift the brain and sever the spinal cord with a slender knife as low down as possible in the foramen magnum. In all cases where disease of the hypophysis is suspected this organ should be carefully removed and fixed for

histological study in 10 per cent formalin, or preferably in Zenker's fluid or formalin-bichromate.

After removal of the brain the dura of the base of the skull should be examined for abnormalities (tubercles, gummata, tumors, etc.). The dura may be torn in fractures of the base of the cranium; such tears can be easily made out. The dural venous sinuses should be explored for thromboses, particularly if the case be one of infection of the air-cells. After this the dura should be stripped from the skull and the cranial base carefully studied for linear fractures. The air-cells (mastoid including middle ear, ethmoid, sphenoid, and frontal) should be opened and examined for infection or other abnormality.

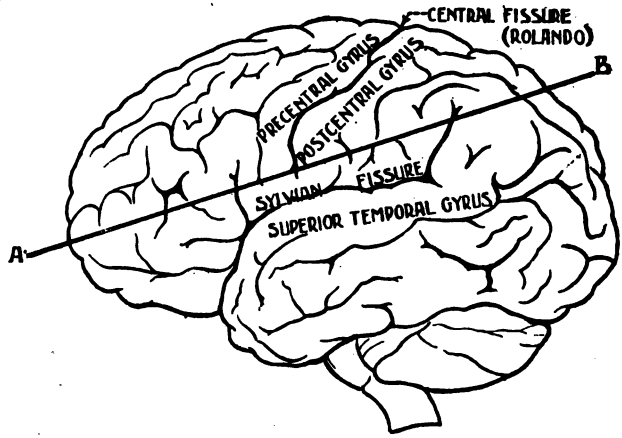


FIG. 216.—Illustration showing line of cut for exposing interior of brain for postmortem examination. (Instruction Laboratory, Army Medical Museum.)

Examination and preservation.—After removal of the brain, the meninges of the floor of the cranium and brain base should be studied as outlined above. By *very gentle* palpation abnormalities of brain consistency may be detected and indications of deep tumors, hemorrhages, or abscesses noted.

The brain should now be weighed and immediately immersed in a liberal amount of 10 per cent formalin. A small amount of sodium chlorid may be added to bring the specific gravity of the fluid up to nearly that of the brain and thus reduce deformation by gravity during hardening. The brain should be supported on a soft cotton pad and turned at intervals of twelve hours for two or three days.

Should it be necessary for medico-legal or other reasons to examine the interior of the brain immediately, it should be opened by a single cut, as follows: With a single sweep of a long, thin autopsy knife cut through both cerebral hemispheres in a plane parallel with and one-half inch above the Sylvian fissures (as indicated on the

sketch, Fig. 216, at A–B). The appearance of this section as seen from above is shown in Figure 217.

It will be noted that this is not the usual horizontal cut passing through the longest axis of the hemisphere, but that the plane inclines downward sharply in front and upward toward the occiput. By this cut the structural relationships of the brain stem are left undisturbed and at the same time the lateral and third ventricles and internal capsule can be explored.

Another procedure which may in many cases be used to advantage is the simple splitting of the corpus callosum. This allows access to

FIG. 217.—Photograph showing cut section of brain after procedure in Fig. 216, looking from above downward. (Instruction Laboratory, Army Medical Museum.)

the third and lateral ventricles and does not cause undue destruction of the brain tissue.

After either exposure, the walls of the cerebral ventricles and the fluid contained therein should be carefully studied for evidences of infection. This usually is shown by a slight loss of normal luster or by roughening of the smooth ventricular wall (best seen by reflected light), and in the more extreme cases, by fibrinous exudate and a purulent cerebrospinal fluid. The choroid plexuses at the same time should be examined; normally of a rich pink color, these change to a brilliant red and become swollen when infected. Study of the ventricle must be emphasized as affording possible evidence of the pathway of infection of the cerebrospinal spaces.

The spinal cord.—In all cases where possible the spinal cord should be examined, and in this event the vertebræ may be examined for development, abnormalities, malpositions, and traumatic conditions. A chisel should not be used in cutting the spinous processes of the vertebræ, but saw and bone forceps only. Before removing, examine the cord in situ and note the amount and distribution of blood in the dura and of the spinal fluid in the dural sac. In removing the cord only gentle traction should be used, as the careful handling of the nerve roots is important, especially in degenerations which have been often simulated by artefacts due to rough handling. Open the dura by slitting it posteriorly with a pair of small scissors, care being taken to raise it during the cutting to avoid injury to the underlying cord.

Gentle palpation may reveal areas of softening or of sclerosis. Areas of softening should not be incised in the fresh state because of inevitable disturbances thus produced in the relations of the component parts. Describe carefully all traumatic lesions.

It is preferable to have the whole cord suspended in 10 per cent formalin with a small weight tied to the lower end of the dura, or the cord may be cut into three pieces and each piece sewed through the dura to a stiff card. When a cord must be sectioned further, due regard should be given to the requirements of further study. Transverse cuts may be made (preferably after fixation) but the attached dura should not be cut through, thus leaving each piece in its proper place in the dural sheath.

Preservation.—Unless a very generous amount of fluid has been used in hardening, the formalin should be renewed or strengthened after two or three days. Each specimen should be tagged by a label cut from sized heavy white cloth tied to the basilar artery of the brain or to the dura of the cord. This should be written upon with waterproof India ink or a *soft* lead pencil and then dipped into melted paraffin. White celluloid tags may be used if available. Paper tags of any kind are worthless, as they soon macerate in the fluid. Labels pasted upon the outside of the container should not in any case be depended upon; even permanent museum preparations should have in addition an identification tag attached to the specimen, and in this case stencilled aluminum tags are permanent in most fluids. The identification tag should carry at least two kinds of marks (preferably the case number and the name of the patient), so that if one is obliterated the other may be used, and full records of the case with similar identification marks should go with the specimen.

Formulæ.—In the following paragraphs are given selected formulæ for reagents most useful for the preservation of gross and microscopic neurological specimens. Ten per cent formalin is unques-

tionably the most generally useful single fluid for this purpose and the simplest to use. It does destroy, however, some of the colors, is only an indifferent cytological fixative, and after long time impairs the staining qualities for general histological purposes.

For **museum specimens** the methods of Kaiserling and Klotz are preferable. The colors of specimens which have been several weeks in formalin can be partly restored by passing them through Kaiserling's fluids. Where both histological and gross preservation are desired, the material may be hardened for two weeks or less in formalin, the pieces desired for histological study then cut out, and the remainder of the specimen passed through the Kaiserling process. Specimens first hardened in formalin and then passed through the Kaiserling process also give good histological sections if skilfully prepared. Material fixed by the Kaiserling method can be used for some histological purposes, though the staining quality is greatly impaired and special methods of intensive staining must be used to get the best results.

Kaiserling's method.—1. Fix in the following solution for one to five days:

Strong formalin (40 per cent formaldehyde)	200 c. c.
Water	1,000 c. c.
Nitrate of potassium	15 gm.
Acetate of potassium	30 gm.

2. Drain and place in 95 per cent alcohol for one to six hours, or until the color is restored.

3. Preserve permanently in:

Acetate of potassium	200 gm.
Glycerin	400 c. c.
Water	2,000 c. c.

Klotz's method.—Make up modified Carlsbad salts in quantity, as follows:

Potassium sulphate	40 gm.
Sodium or potassium nitrate	760 gm.
Sodium chloride	360 gm.
Sodium bicarbonate	400 gm.
Sodium sulphate	440 gm.

FLUID No. 1.

Carlsbad salts	375 gm.
Chloral hydrate	375 gm.
Formalin	375 c. c.
Water	15 liters.

FLUID No. 2.

Carlsbad salts	375 gm.
Chloral hydrate	150 gm.
Formalin	75 c. c.
Water	15 liters.

Fix three to five days or longer in fluid No. 1. Wash three to five hours in running water. Preserve in fluid No. 2.

The following fluids are to be used only for small pieces of tissue for histological purposes:

FORMALIN-BICHROMATE.

To nine parts of a 2½ per cent solution of potassium bichromate in water, add one part of strong formalin (40 per cent formaldehyde). This solution should be freshly made just before using and if used for more than twenty-four hours renewed daily. Small pieces will be fixed in twelve hours or less and when so used the fluid is especially valuable for cytoplasmic fixation. For Weigert preparations it should be used from three to five days, after which the tissue should be washed thoroughly and either dehydrated and embedded immediately or left in 10 per cent formalin for storage.

ORTH'S FLUID.

This is Müller's fluid, to which 10 per cent formalin is added just before using. It is used the same as formalin-bichromate. Müller's fluid alone for several weeks or Orth's fluid for a few days are most commonly used fixing fluids for Weigert preparations, though excellent Weigert preparations (and also Marchi preparations) can be made from formalized material.

MÜLLER'S FLUID.

Potassium bichromate	25 gm.
Sodium sulphate	10 gm.
Water	1,000 c. c.

ZENKER'S FLUID.

Potassium bichromate	2. 5 gm
Mercuric chloride	5. gm.
Sodium sulphate	1. gm.
Water	100. c. c.

Just before using add 5 c. c. glacial acetic acid or 10 c. c. strong formalin.

This is one of the most widely used general histological fixing fluids. Small pieces, not over 5 mm. in thickness, must be used and fixation should usually not exceed twelve hours, sometimes less. Wash thoroughly in water before dehydration and the mercury must be extracted by adding iodin to the alcohols during dehydration or subsequently. The formula with formalin gives better cytoplasmic fixation than that with acetic acid, though each has its special advantages.

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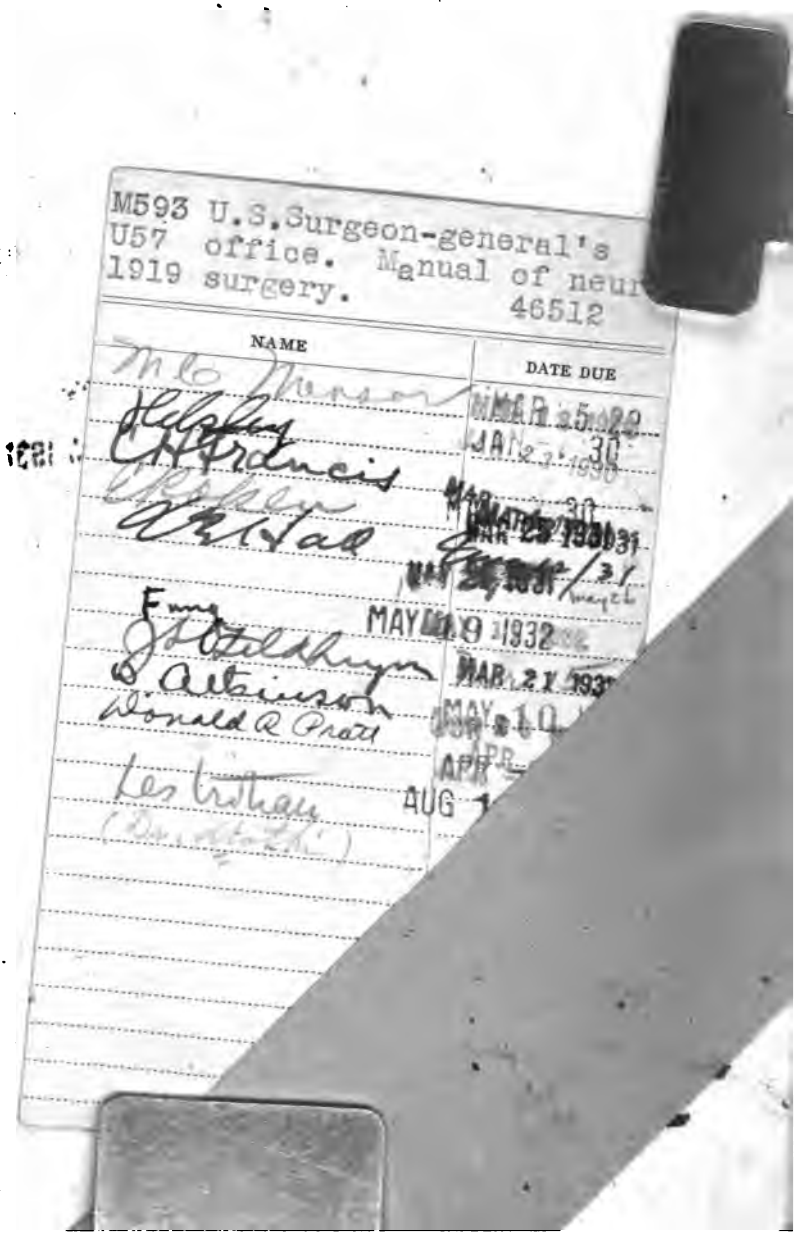
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